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THE MOTOR ACTIVITY OF THE CRICOPHARYNGEUS MUSCLE.*†‡**

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Of the many muscles involved in the complex act of swallowing, none has equaled the cricopharyngeus in stimulating the curiosity of the investigator, the interest of the clinician, and the respect of the esophagoscopist.

First mentioned as a distinct anatomical entity by Valsalva[1] in 1717, the cricopharyngeus was recognized by succeeding anatomists,[2,3,4] who usually included it with the inferior constrictor muscle, as its lower-most or cricopharyngeal portion. Its sphincter-like action was recognized in 1823 by Home[5] and later discussed by Mikulicz,[6] 1881; Killian,[7] 1908; and Jackson[8] in 1914. Its relation to the mouth of the esophagus was carefully studied by Elze[9] in 1929, and more recently by Lerche.[10]

Its physiological identity has been further demonstrated by studies in comparative[11] and gross anatomy,[12] by roentgenographic demonstration of sphincter action,[13,14] and by the relief of post-poliomyelitic dysphagia following surgical section of the cricopharyngeus muscle.[15]

Spasm of the cricopharyngeus muscle and its production by

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autonomic imbalance is frequently mentioned by clinical authors as a cause of dysphagia, but without much experimental evidence to support it. One of the few basic investigations of this problem was reported by Sjöberg in 1939,[16] involving fluoroscopic studies of the barium-filled esophagus in cats after various combinations of cranial nerve sections in the neck. After bilateral vagotomy, autopsy showed a narrowed condition in both the upper and lower ends of the esophagus, attributed by Sjöberg to spastic contractions during life. Administration of ergotamine tartrate to vagotomized cats to combat sympathetic activity produced relaxation at both the cricopharyngeal and cardiac ends of the esophagus. The parasympathetic action was then simulated in these animals by injection of acetyl-choline, whereupon the cricopharyngeus relaxed and peristaltic waves were seen passing down the esophagus. Sjöberg's conclusion from these findings was that the esophageal ostia are caused to contract by the adrenergic sympathetic fibers, while the cholinergic vagus fibers bring about relaxation.

Further support for this interpretation was furnished by Rogers,[17] who reported relief of severe dysphagia in a woman with a spastic cricopharyngeus muscle on whom he performed bilateral extirpation of the superior cervical sympathetic ganglia. One must admire his courage as well as his surgical result, for his operation had very little experimental or clinical evidence to recommend it. Even today a sympathetic supply to the cricopharyngeus is flatly denied by Hollinshead,[18] who states that "the cricopharyngeus muscle, being a voluntary one, is not innervated by the sympathetic system, and there seems to be no ready explanation as to why a cervical sympathectomy should relieve spasm of this muscle, as it was reported to do by Rogers."

Although the main vagal branches to the cricopharyngeus have been identified,[19] and a sympathetic as well as a parasympathetic component is generally agreed to exist, there is no definite mention in modern literature as to what type of activity (contraction or relaxation) is mediated by the vagus or sympathetic branches to the cricopharyngeus.[20,21,22] Nor is it even certain that a specific type of activity is mediated

exclusively by one or the other autonomic division as shown by studies on the cardiac end of the esophagus. Carlson, for example, showed that the vagus carries both motor and inhibitory fibers to the cardia, and that the splanchnics (sympathetic) also carry both motor and inhibitory fibers to this area, the action depending on the state of contraction of the cardia at the time of stimulation.[23]

The purpose of my present study, therefore, is to demonstrate, in each case, the effect of stimulation or interruption of the cervical sympathetic or parasympathetic trunks on the motor activity of the cricopharyngeus muscle.

Such experimental conditions simulate the autonomic imbalance said to exist in certain cases of spastic dysphagia. The resulting increase or decrease in tonus can then be demonstrated by means of a pressure-sensitive recording device placed in the hypopharynx at the cricopharyngeus level.

Pharyngeal pressures during deglutition have been studied and recorded by investigators since the late Nineteenth Century. In 1880 Falk and Kronecker measured the pharyngeal pressure generated during deglutition in themselves and in dogs. They observed the "squirting" action on liquids and semi-solids exerted by the muscles of the mouth and the pharynx by the use of a "T" tube.[24] Kronecker and Meltzer in 1883 reported their observations of the swallowing mechanism in dogs, using small balloons to record intraesophageal motility.[25] Meltzer considered the squirting action to be the result of pressure exerted by the mylohyoid muscles, because he was able to eliminate it by sectioning the mylohyoid nerves. He reported that this operation made the first stage of swallowing impossible, so that the bolus had to be introduced by the operator into the dog's pharynx in order for the animal to swallow.[26] This same investigator, in 1907, added further evidence of the importance of the oral musculature in swallowing by cutting the middle and inferior constrictors of the pharynx in the dog, in addition to removing the muscularis of the cervical esophagus. A dog, prepared in this fashion, was still able to swallow normally and could drink from a bowl of milk placed below the level of the head, thus eliminating

the effect of gravity.[27] Cannon and Moser, in 1898, were the first to use fluoroscopy to study esophageal motility in animals. These workers also noted the rapid squirting action of fluids from the pharynx to the level of the heart.[28]

Although these experiments contributed much to our knowledge of the motor activity of the pharyngeal and oral musculature, the graphic recording of such motor activity was inexact, and the pressure readings unreliable.[29] It was not until 1943, with the development of an electronic device by Wetterer,[30] that a practical means of recording intraluminal pressures was made available. Wetterer described the principle of using a differential transformer to construct a miniature manometer, a system having excellent recording properties with a minimum of amplification. The device was later modified by Gauer and Gienapp[31,32] in 1950 to fit into the end of a No. 8 Cournand catheter for the recording of intracardiac and intravascular pressures. This instrument was used by Fyke and Code in 1955 for recording pressure changes in the pharyngoesophageal region during deglutition in normal human subjects.[33] Their work showed a definite pressure gradient beginning in the pharynx and traveling down through the cricopharyngeus area into the esophagus during the act of swallowing. The high pressure in the cricopharyngeus region remained unchanged throughout most of the act of swallowing, and dropped only momentarily when the pressure wave reached this area.

The activity of the individual musculature involved in swallowing was later studied by Doty and Bosma by means of electromyography. These workers took electromyographic records from 22 muscles likely to participate in deglutition. Their work substantiated that of Fyke and Code, showing a downward progressing contraction wave and an inhibition of the inferior constrictor during the entire course of lead muscle activity.[34]

PRESENT STUDY.

After controls had been established in the normal dog, the action of the cricopharyngeus muscle was studied under the following conditions:

1. Unilateral vagus section above the ganglion nodosum.

2. Bilateral vagus section above the ganglion nodosum.

3. Faradic stimulation of the vagus and cervical sympathetic trunks.

4. Faradic stimulation of the cricopharyngeus muscle.

5. Surgical section of the cricopharyngeus muscle.

The methods used in studying the motor activity of the cricopharyngeus were the following:

1. Observation of the dog's ability to swallow his usual food.

2. Endoscopic examination of the soft palate, pyriform sinuses and cricopharyngeus sphincter area with the dog under light general anesthesia.

3. Fluoroscopic and roentgenographic studies of the hypopharynx with barium mixtures.

4. Pressure changes in the hypopharynx at the cricopharyngeus level during deglutition, using an electronic pressure-sensitive pick-up.

These methods are standard and seem self-explanatory except for the last, which requires further explanation.

In the beginning of this experiment we were using a Gauer-type pressure capsule attached to the proximal end of a 2 mm. polyethylene tube introduced through the mouth to the cricopharyngeus level, and recording the motor activity of the cricopharyngeus by means of a Hathaway oscillograph. This gave satisfactory and sensitive responses, as shown by Fyke and Code, but seemed to have certain disadvantages. First, if a fold of mucosa came to overlie the end opening (or side opening as was later tried) the pressure determination was interfered with; therefore, the open end was enclosed in a small, partially inflated balloon. This, however, was soon abandoned because of distortions produced in the balloon by the contracting cricopharyngeus muscle, with resultant inaccuracies in the reproduction of intraluminal pressures.

My chief objection, however, to present techniques of re-

cording pressures at the cricopharyngeus level is that this area is pulled upward during swallowing, so that a recording device introduced to the level of the cricopharyngeus sphincter at rest and kept at this level during swallowing is recording pressure not in the cricopharyngeus segment but in the upper cervical esophagus. While there is some evidence that a small polyethylene tube will ride up and down with the cricopharyngeus[35] during deglutition, there seemed enough doubt

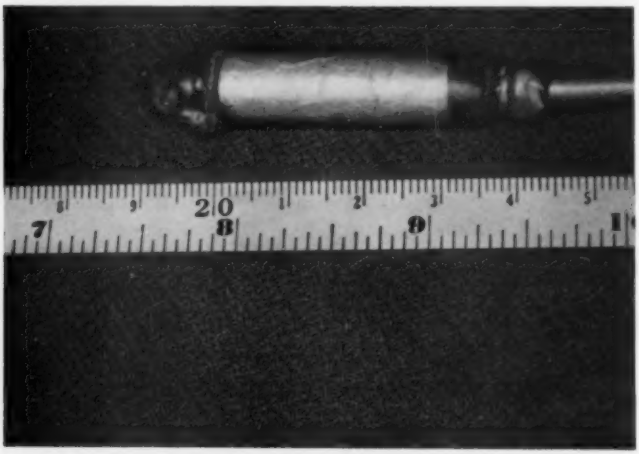


Fig. 1. Pressure-sensitive electronic pick-up used in hypopharynx at cricopharyngeus level.

on this point to have warranted the construction of a pressure-recording device which would respond with equal sensitivity along a 3 or 4 cm. distance. If such a device were of small diameter and of smooth surface it could be introduced into the pharyngo-esophageal junction behind the cricoid cartilage, allowing the cricoid and cricopharyngeus to ride up and down over it, and recording the motor activity of the cricopharyngeus whether it was elevated or at rest.

For the development of such a recording device I am indebted to Dr. Albert Field, Jr., of the Cardiopulmonary section of the Department of Internal Medicine. A pressure-sensitive

Fig. 2. Dog No. 1—Normal. AP view after barium swallow. The trachea deviates to right in supine position in this case, but may deviate to left, as seen in other examinations.

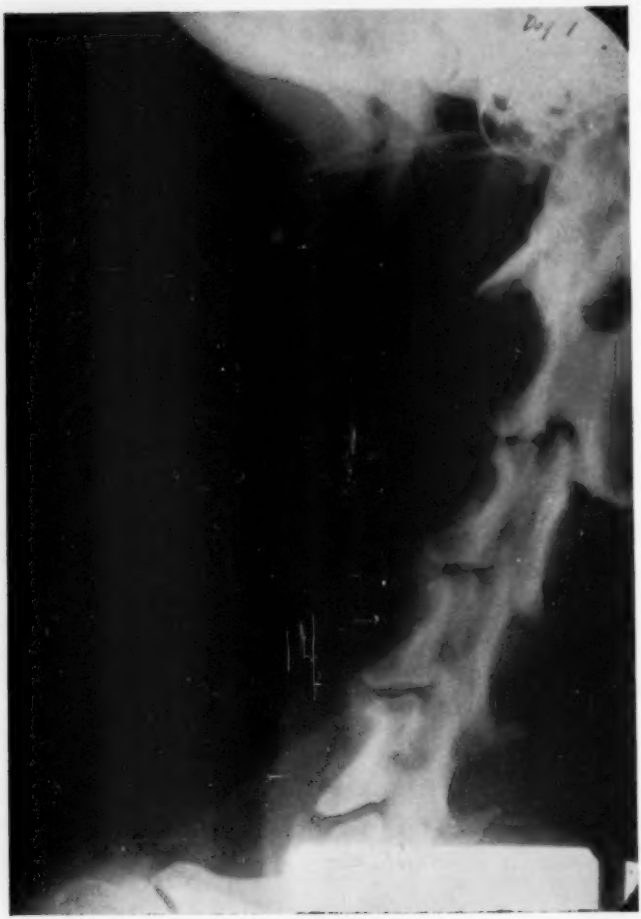


Fig. 3. Dog No. 1—Normal lateral view after barium swallow. A very small amount of barium remains in the valleculae.

pick-up was constructed, consisting of a variable capacitance which measures pressure changes by means of variations in a tuned circuit, based on a principle described by Buchtal and Warburg in 1943.[36] It is equally sensitive to pressure at any point along the distance of 3.5 cm., and allows a permanent record to be made by a direct writing electrocardiographic instrument.

The equipment has been checked for performance against a Sanborn pressure capsule and found to be accurate, sensitive

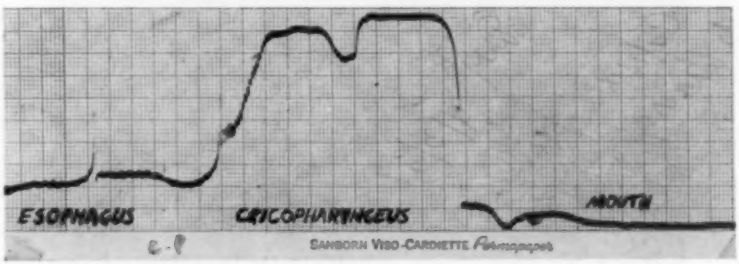


Fig. 4. High-pressure area encountered at cricopharyngeus as cable is withdrawn from thoracic esophagus.

and brisk in response. Calibrations and other details are described in a separate report.

Because of variations in the base line under different operating conditions, calibrations for exact pressures are not reliable. The calibrations are included, nevertheless, (mm. Hg.) to indicate direction of deflections with positive and negative pressure and to indicate approximate pressure ranges. Changes in pressure are, of course, readily demonstrated by the shape of the curves.

NORMAL DOG.

Deglutition: Ate regular diet without difficulty.

Endoscopic Examination: The soft palate, pharynx, larynx and tongue move normally. A definite annular contraction was seen behind the cricoid cartilage.

X-ray: Fluoroscopy of a thin barium mixture showed rapid descent of the liquid from the mouth to the level of the heart with no tendency to collect in any of the pharyngeal recesses. A normal soft tissue pattern behind the cricoid cartilage was seen on the lateral views (see Figs. 2, 3).

Pressure Studies: Because of the fact that deep general anesthesia abolishes the swallowing reflex, light nembutal

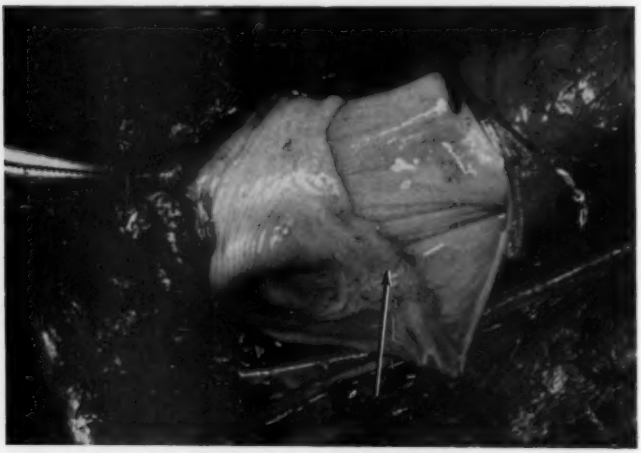


Fig. 5. Dissected specimen showing annular fold in hypopharynx behind cricoid cartilage.

anesthesia was used. Pressure readings were made when the deglutition reflex could be stimulated by gently stroking the posterior pharyngeal wall.

The pressure capsule was first introduced into the thoracic portion of the esophagus and the recording base line adjusted on the recorder. The capsule was then slowly pulled up from the esophagus into the mouth, during which an area of high pressure was encountered in the cricopharyngeal region (see Fig. 4). This was a constant finding, whether the dog was in a prone or supine position. This high pressure area was found to be 19.5 cm. from the upper incisor teeth and corre-

Fig. 6. Dissected specimen showing pressure pick-up in position at cricopharyngeus level.

Fig. 7. Dissected specimen showing cricopharyngeus and thyropharyngeus portions of inferior constrictor muscles. (Operator's finger is in lumen of hypopharynx.)

sponded almost exactly to the area of annular constriction seen on endoscopic examination (see Fig. 5). The mid-point of the recording capsule was introduced to this level for deglutition patterns and other studies in this dog (see Fig. 6).

The swallowing patterns shown here illustrate the characteristic curve obtained in all our experimental animals. At the beginning of cricopharyngeal activity a small rise in

Fig. 8. Dissected specimen showing cricopharyngeus portion (arrow) of inferior constrictor separated from thyropharyngeus. Pressure determinations were made at cricopharyngeus level.

pressure can be seen, probably corresponding to the advancing peristaltic wave. Almost immediately the cricopharyngeus undergoes a marked relaxation, followed again by a contraction, which may rise to or above the original base line level (see Fig. 9).

SECTION OF ONE VAGUS NERVE.

In the dog the vagus nerve carries with it the sympathetic trunk throughout most of its cervical course. The sympathetic joins the vagus opposite the inferior cervical sympathetic ganglion and leaves it again just caudal to the ganglion

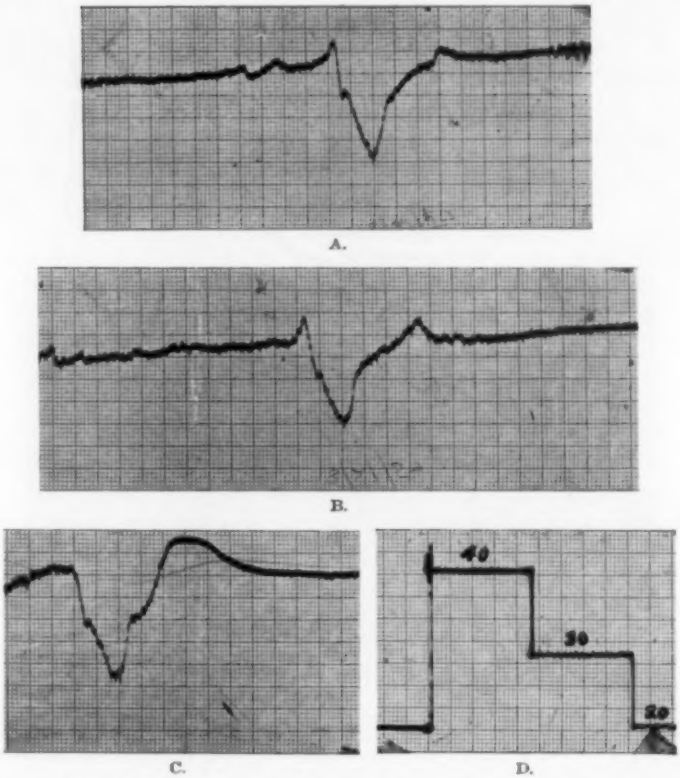


Fig. 9. A.-B.-C.—Pressure recordings from cricopharyngeus level during act of swallowing. D.—Calibration (mm. Hg.).

nodosum, where it joins the superior cervical sympathetic ganglion. The pharyngeal ramus of the vagus, which supplies parasympathetic innervation to the constrictor muscles leaves the vagus trunk cephalic to the ganglion nodosum at its point of emergence from the jugular foramen. Whether the sympathetic trunk gives off separate rami to the pharyngeal plexus or to the pharyngeal musculature along its cervical course, as it does in man, I could not determine from the available literature (see Fig. 10 for details).

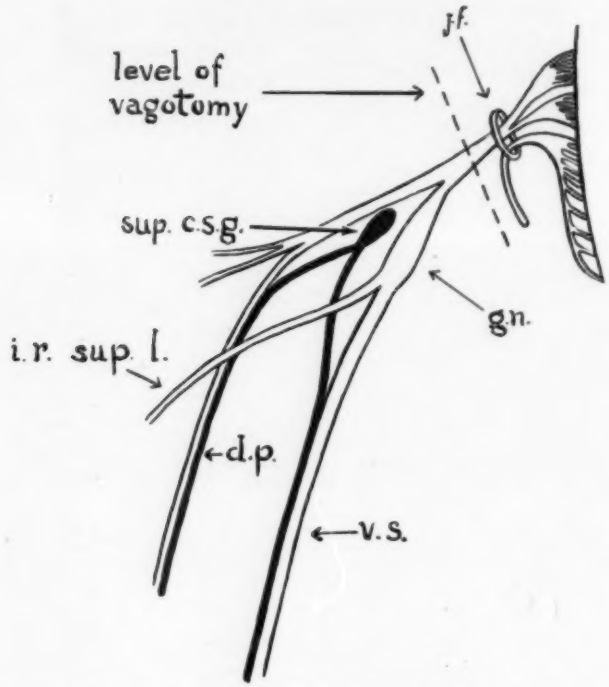


Fig. 10. Diagram showing trunks of laryngeal and pharyngeal nerves (from Lemere[19]. j.f.—Jugular foramen; g.n.—Ganglion nodosum; v.s.—Vago-sympathetic trunk; d.p.—descending pharyngeal; i.r. sup. l—internal ramus of superior laryngeal; sup. c.s.g.—superior cervical sympathetic ganglion. Level of vagotomy shown by dotted line.

The right vagus nerve was exposed by dividing the muscle which corresponds in the dog to the digastric, then sectioned at its emergence from the base of the skull in order to include the pharyngeal ramus (see Fig. 11).

Deglutition: The dog continued to eat normally during the entire period of observation of three weeks.

Endoscopic Examination: The right vocal cord remained motionless in a partially abducted or cadaveric position. There was a curtain movement of the pharynx to the left and a deviation of the soft palate to the left.

X-ray: The barium mixture cleared the pharynx easily. There was no residual barium in any of the pharyngeal recesses nor were there any changes in the soft tissue patterns (see Figs. 12, 13).

Pressure Studies: Swallowing patterns show much less relaxation of the cricopharyngeus during swallowing, with a higher positive pressure (see Figs. 14, 15).

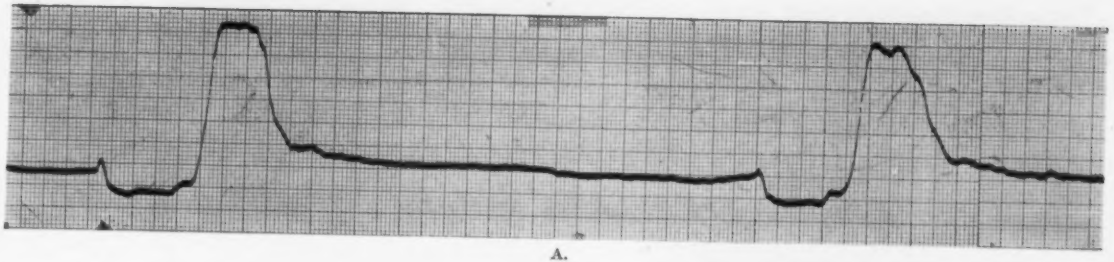
Fig. 11. Vagus nerve (arrow) approaching base of skull.

DISCUSSION.

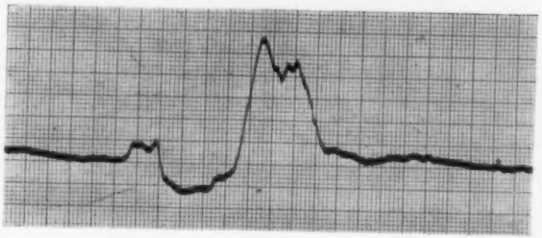
It is interesting that this dog had no dysphagia, although there was a curtain movement of the pharynx, deviation of the soft palate, and paralysis of the right vocal cord. This may be due to the fact that the free portion of the epiglottis of the dog lies above the soft palate, allowing the ingested food and liquids to pass down lateral to the larynx and affording added protection to the laryngeal inlet during the act of swallowing. The decreased relaxation of the cricopharyngeus during deglutition following unilateral vagus section may be due to an over-activity of the remaining sympathetic nerve supply. I do not believe it is due to a lower starting pressure,

Fig. 12. Dog No. 1—Two weeks post-vagotomy, right. Barium swallow shows no essential change from unoperated condition.

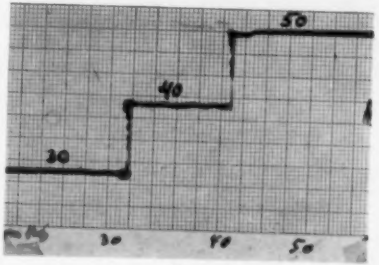
Fig. 13. Dog No. 1—Two weeks post-vagotomy, right. No essential change from unoperated condition.



A.



B.



C.

Fig. 14. A., B.—Pressure recording of deglutition immediately after unilateral vagotomy. C.—Calibration. The relaxation phase is much less prominent than before operation.

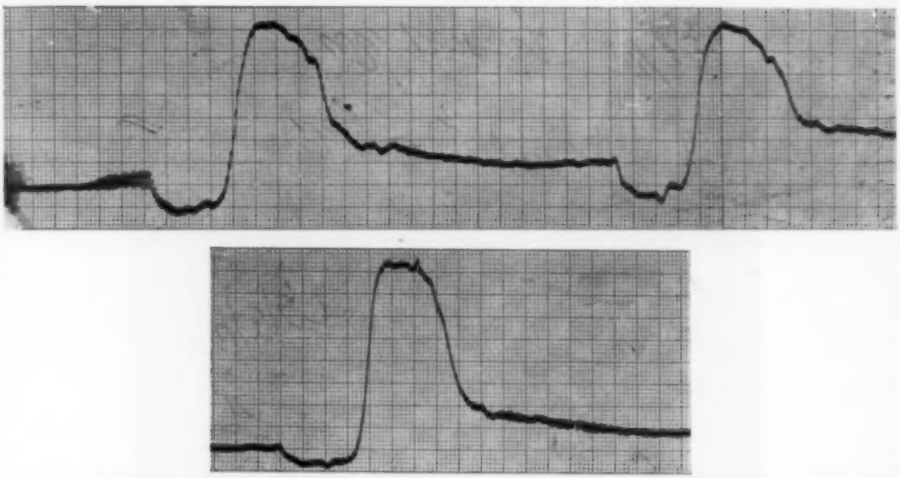


Fig. 15. Pressure recordings during deglutition ten days after unilateral vagotomy. Shows minimal relaxation.

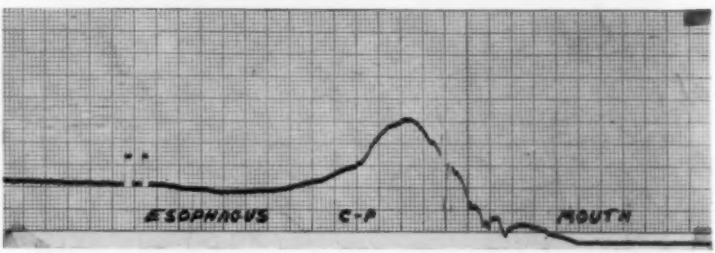


Fig. 16. High-pressure area at cricopharyngeus after unilateral vagotomy. No essential change from pre-operative condition.

since the pressure capsule when pulled up through this area from the esophagus showed the usual pressure rise when passing the cricopharyngeus (see Fig. 16).

SECTION OF BOTH VAGI.

Using the original dog from the first experiment, the left vagus was divided at its point of exit at the base of the skull three weeks after section of the opposite vagus.

Fig. 17. Dog No. 1, 24 hours after division of second vagus. Barium lies in valleculae and in trachea.

Fig. 18. Dog No. 1, 24 hours after division of second vagus. Barium lies in valleculae, pharynx, upper esophagus and trachea.

Deglutition: Following recovery from anesthesia, the dog exhibited obvious dysphagia, throwing his head upward and backward in an attempt to swallow liquid.

Endoscopic Examination: Both vocal cords were paralyzed in a paramedian position. Saliva was collected in the lower recesses of the pharynx.

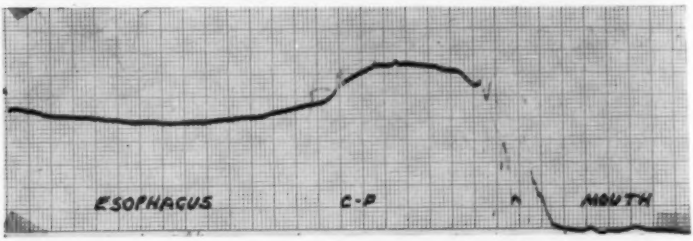


Fig. 19. High-pressure area at cricopharyngeus following bilateral vagotomy.

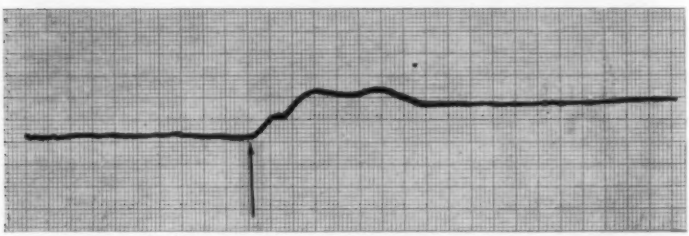


Fig. 20. Pressure rise at cricopharyngeus at moment of section of second vagus. This and the following two figures are tracings of technically unsatisfactory originals.

X-ray: Barium mixture was found puddled throughout the pharynx, with some aspiration into the trachea. Lateral views showed no significant changes in the soft tissue patterns on the posterior or anterior walls (see Figs. 17, 18).

Pressure Studies: Before the second vagus was cut, the cable was pulled up from the esophagus through the cricopharyngeus (see Fig. 19). The pressure pick-up was then placed in the cricopharyngeus area and a recording taken at

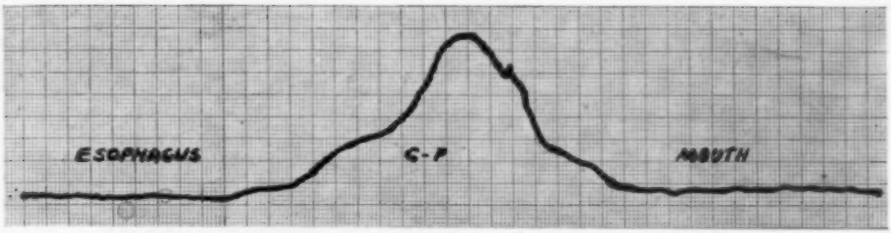
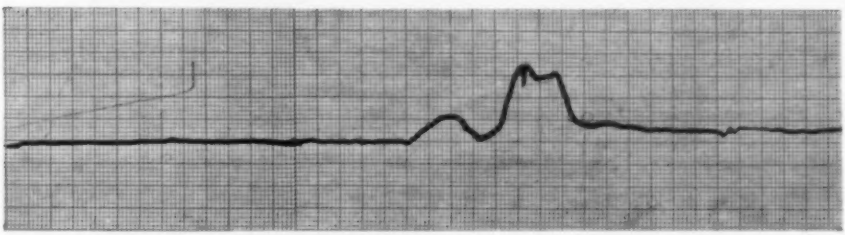
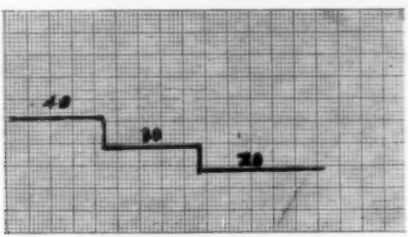


Fig. 21. Tracing of original recording of high-pressure area at cricopharyngeus following bilateral vagotomy.



A.



B.

Fig. 22. A.—Tracing of original recording of deglutition pattern following bilateral vagotomy. Relaxation phase is absent. B.—Calibration.

the moment the second vagus was sectioned (see Fig. 20). There was a slight, but definite increase in pressure at the time the vagus was sectioned. The cable was then reinserted into the thoracic esophagus, slowly pulled upward through the cricopharyngeus area, where the high-pressure area was encountered as in the previous experiments (see Fig. 21).

Swallow patterns showed complete absence of relaxation of the cricopharyngeus during the act of deglutition (see Fig. 22).

DISCUSSION.

Section of both vagi produces a condition in which the cricopharyngeus does not relax during the act of swallowing. This is probably due to a relative overactivity of the sympathetic supply, and probably explains the dysphagia resulting from nuclear or high cervical lesions of the vagus nerves. Paralysis of the remaining constrictor muscles resulting from such lesions undoubtedly contributes to the dysphagia, but does not, in itself, make swallowing impossible, as shown by Meltzer, in cutting the middle and inferior constrictor muscles.[27] The real blockade is produced by the tight cricopharyngeus, whose failure of relaxation, as shown in Fig. 22, suggests achalasia as it is encountered at the lower esophageal ostium.

STIMULATION OF THE VAGUS AND SYMPATHETIC.

In order to eliminate any possible pressures resulting from the rocking of the thyroid on the cricoid cartilages by the cricothyroid muscle during this experiment, the cricothyroideus was first divided bilaterally in a normal dog. The vagus nerve was exposed near the base of the skull after dividing the digastric muscle so that the stimulating electrode could be placed between the ganglion nodosum and the jugular foramen. The dog was lightly anesthetized with 5 per cent nembutal.

X-ray: Barium mixture was inserted into the dog's pharynx and the dog made to swallow by stroking the posterior wall of the pharynx. The right vagus nerve was then stimulated, but inspection of the neck after the films were taken showed that the electrode had been pulled downward along the vagus so that the entire vagosympathetic trunk had been stimulated. AP views show a marked deviation of the trachea to the left, away from the side of vagal stimulation, as one would expect, with relaxation of the supporting musculature on one side. While no conclusions are drawn from this finding because a slight tracheal deviation was found pre-operatively, the finding, nevertheless, warrants further investigation (see Figs. 23, 24).

Fig. 23. Stimulation of right vagus, dog No. 2. Tracheal deviation to opposite side is suggestive of relaxation of right constrictor muscle support, but inconclusive.

Fig. 24. Stimulation of right vagus, dog No. 2. No significant findings.

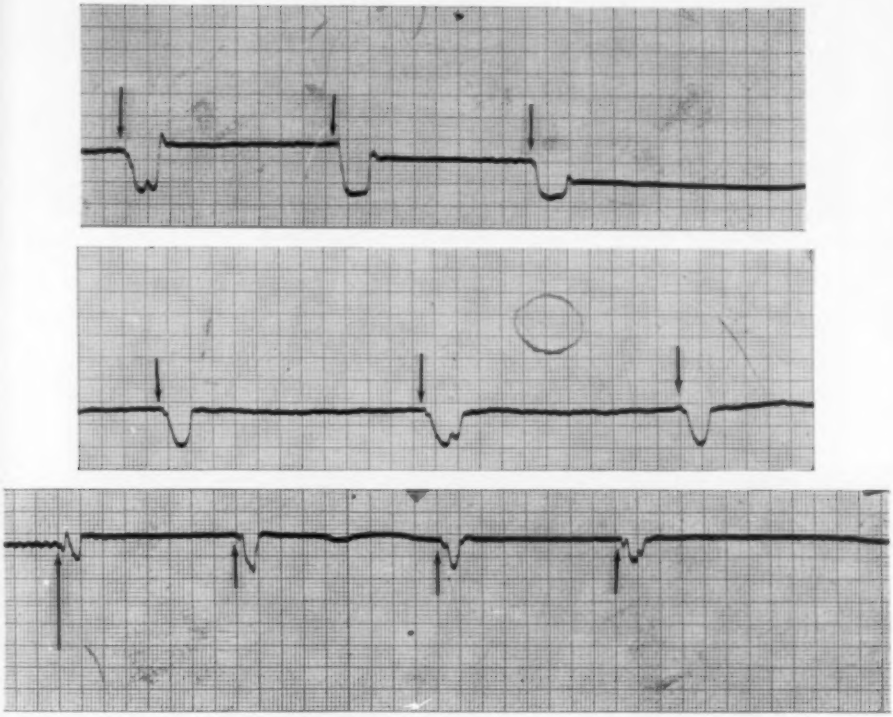


Fig. 25. Faradic stimulations (arrows) of right vagus. Sharp relaxation of cricopharyngeus is shown.

Pressure Studies: The faradic stimulus was adjusted to a low intensity so that vagus stimulation did not produce a swallow or cause noticeable movements of the regional muscles. For sympathetic stimulation, it was assumed that the supply to the cricopharyngeus comes from the superior cervical sympathetic ganglion.[17] Stimulation of the vagus above the ganglion nodosum produced a sudden sharp drop in the resting pressure of the cricopharyngeus muscle (see Fig. 25).

Stimulation at the superior cervical sympathetic ganglion produced a sharp rise in pressure in the cricopharyngeus zone (see Fig. 26). Controls were run with finger pressure over

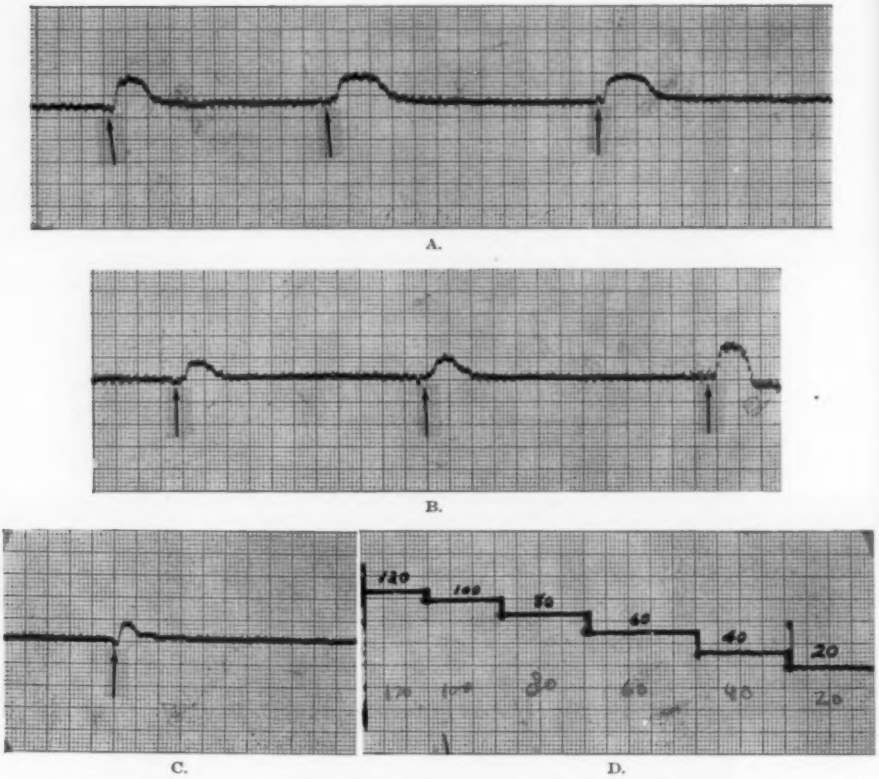


Fig. 26. A., B., C.—Faradic stimulation (arrows) of superior cervical sympathetic ganglion. Definite pressure increases are shown. D.—Calibration.

the cricoid cartilage, producing positive deflections (see Fig. 27). The faradic stimulator was held near, but not on the nerve, to determine changes due to electrical conditions (see Fig. 28). There were none found. The stimulator was also applied to the tongue in order to check the effect of movements of adjacent organs. There were no significant effects noted (see Fig. 29).

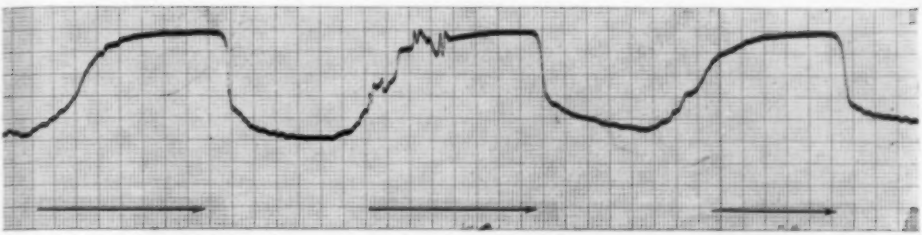


Fig. 27. Control: finger pressure on cricoid cartilage showing upward deflection with increase in pressure (arrows).

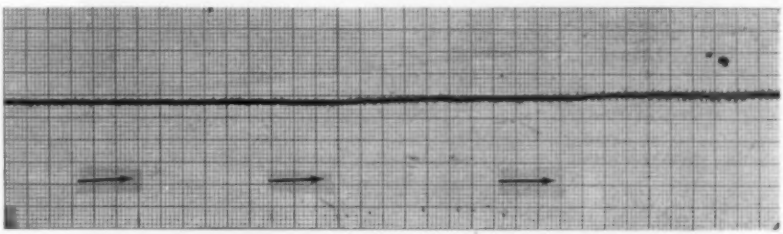


Fig. 28. Control: Electrode near, but not on the nerve.

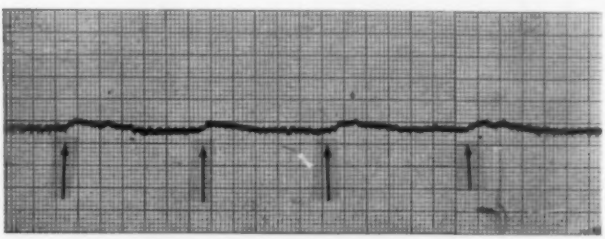


Fig. 29. Control: Electrode stimulating tongue. No significant pressure changes at cricopharyngeus.

DISCUSSION.

The consistent drop in pressure with vagal stimulation, and the consistent rise in pressure with sympathetic stimulation indicates that only one type of activity is mediated by each of the two autonomic divisions. It adds further support to Sjöberg's impression that the sphincteric closure at the crico-

Fig. 30. Faradic stimulation of cricopharyngeus muscle. Less tracheal deviation, but no significant changes.

Fig. 31. Faradic stimulation of cricopharyngeus. No significant changes.

pharyngeus following bilateral vagotomy is due to unopposed sympathetic activity, and that this condition may occur clinically.

STIMULATION OF THE CRICOPHARYNGEUS.

Stimulation of the cricopharyngeus muscle itself produced no appreciable change on X-ray examination from the normal resting stage. There appeared to be a slight tilt of the thyroid

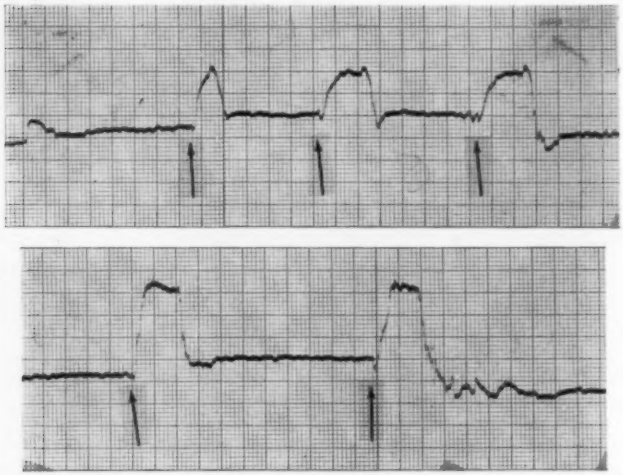


Fig. 32. Faradic stimulation of cricopharyngeus muscle. Sharp increase in pressure.

cartilage on the cricoid, but it is not definite enough to be conclusive (see Figs. 30, 31).

Pressure Studies: Pressure readings during stimulation of the cricopharyngeus muscle show definite contractions with each stimulus (see Fig. 32).

SECTION OF THE CRICOPHARYNGEUS.

In order to determine whether the thyropharyngeus portion of the inferior constrictor has any tonic action in holding the

larynx back against the vertebrae the cricopharyngeus action was eliminated by dissecting the cricopharyngeus off both sides of the cricoid at its points of attachment.

Deglutition: The dog exhibited no dysphagia over an observation period of one month. The right vocal cord was found to be abducted immediately postoperatively, but its action had returned to normal when it was examined about three weeks later.

Endoscopic Examination: There was no collection of food or saliva anywhere in the pharynx, and the annular area noted in the normal dog was found again to be present behind the cricoid cartilage.

X-ray: Swallowing performed normally. No changes in the soft tissue pattern of the pharynx or hypopharynx. There is no change in the position of the larynx as compared to its distance from the posterior wall (see Figs. 33, 34).

Pressure Studies: Faradic stimulation of the vagus supply to the inferior constrictor muscle produced only a barely noticeable drop from resting pressure (see Fig. 35).

DISCUSSION.

Complete separation of the cricopharyngeus from the cricoid cartilage is compatible with normal deglutition. Endoscopic examination shows the annular constriction found prior to surgery. Photomicrographs prepared from this area show that the ridge is composed chiefly of mucous glands (see Figs. 36, 37, 38). Stimulation of the vagus after sectioning the cricopharyngeus produced practically no change in the resting pressure in this area, showing that the upper or thyropharyngeus portion of the inferior constrictor has little or no tonic action in supporting the larynx. Comparison of pressure readings before and after sectioning the cricopharyngeus shows that this muscle is normally in a state of tonic contraction.

SUMMARY.

1. The motor activity of the dog's cricopharyngeus muscle was studied during its resting state and during deglutition.

Fig. 33. Following section of cricopharyngeus and thyropharyngeus, bilaterally. No significant changes.

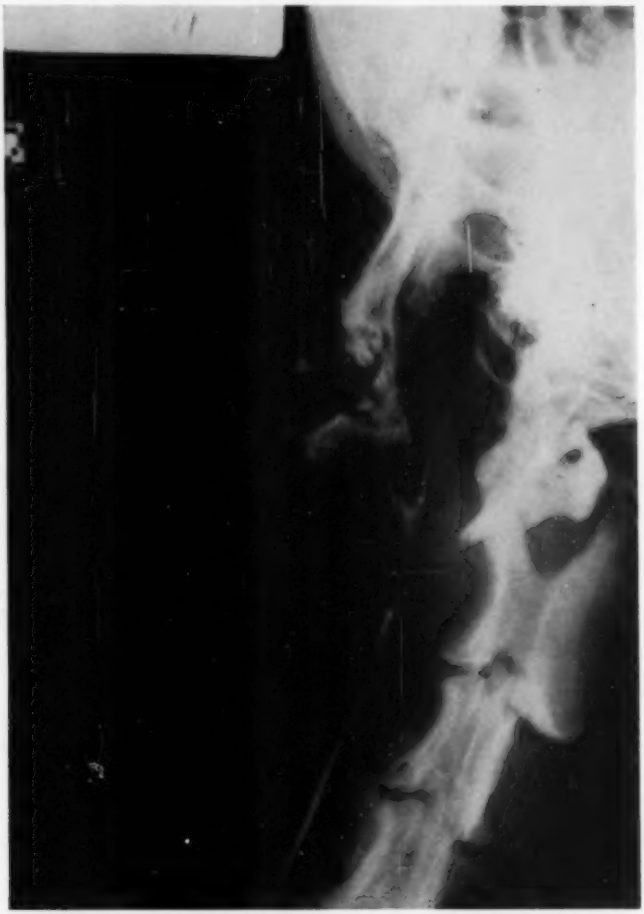


Fig. 34. Following section of cricopharyngeus and thyropharyngeus. No significant changes.

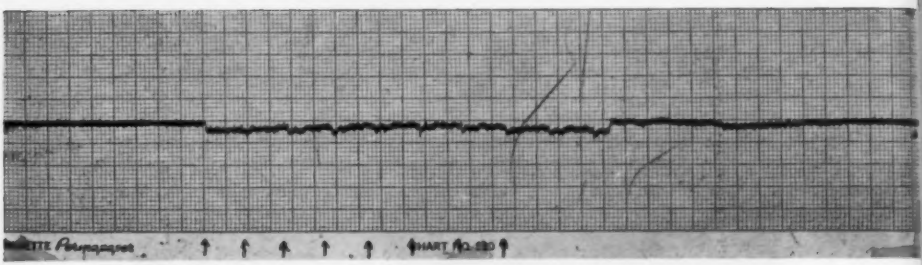


Fig. 35. Faradic stimulation of vagus following section of cricopharyngeus muscle. The usual drop in pressure is not seen.

Fig. 36. Saggital section of wall of hypopharynx at level of cricopharyngeus (arrow) and annular fold (X12). See Fig. 5.

2. By sectioning or stimulating each of the two major components of the nerve supply to the cricopharyngeus muscle various states of autonomic imbalance were produced and their effects observed by four methods:

A. Observations of the dog's ability to swallow food and liquids.

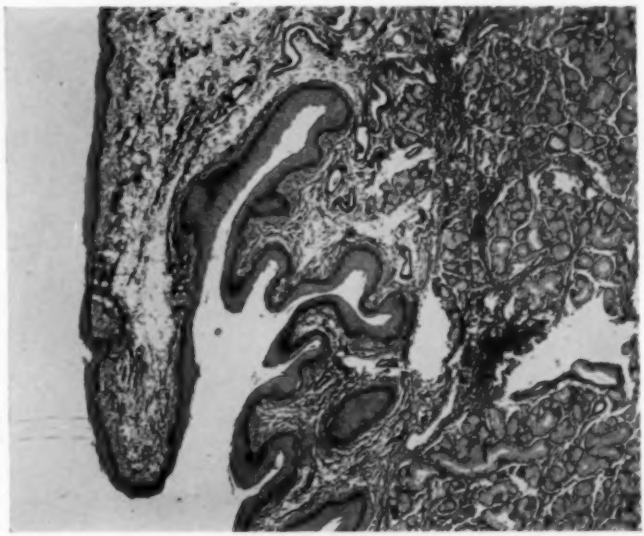


Fig. 37. Saggital section through hypopharyngeal fold (upper square in Fig. 36). (X38).

B. Endoscopic appearance of the pharynx, larynx and hypopharynx.

C. Radiologic studies of the hypopharynx.

D. Intraluminal pressure recordings from the cricopharyngeus sphincter area using a newly developed electronic cable.

3. Controls were established for the pressure recordings by the following means:

A. Manual pressure on the cricoid cartilage with the recording capsule in position.

B. Faradic stimulation of adjacent muscles.

C. Application of the stimulator near to but not on the nerves in order to determine changes due to electrical conditions.

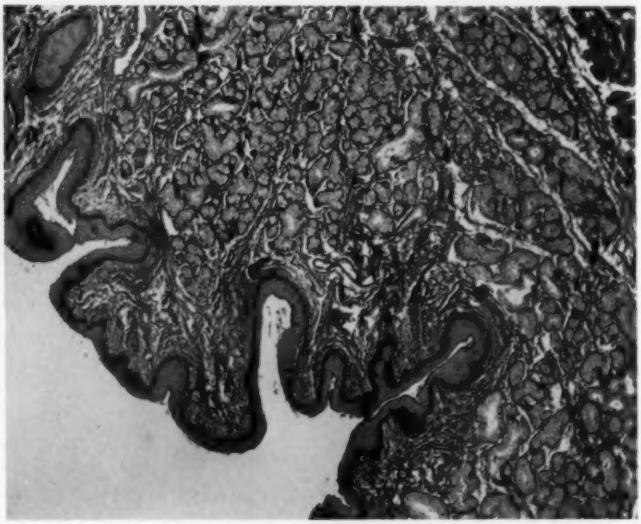


Fig. 38. Saggital section through hypopharyngeal fold (lower square in Fig. 36), showing large masses of mucous glands. (X38).

RESULTS AND CONCLUSIONS.

1. The cricopharyngeus muscle was found by pressure recordings to be in a constant state of tonus during its resting stage.

2. During normal deglutition the muscle first relaxes then contracts to a pressure level equal to or higher than its resting pressure.

3. Unilateral vagus section at base of skull diminishes

slightly the relaxation phase of the muscle during swallowing but does not produce dysphagia in the dog.

4. Bilateral vagus section at base of skull abolishes the relaxation phase and produces severe dysphagia during the dog's remaining hours of life.

5. Faradic stimulation of the vagus branch to the cricopharyngeus produces a sharp relaxation of pressure.

6. Stimulation of the superior cervical sympathetic ganglion produces a sharp rise in pressure.

7. Relaxation is mediated by the parasympathetic fibers; contraction is mediated by the sympathetic.

8. Dysphagia may result from increased tonus in the cricopharyngeus as a result of sympathetic overactivity in lesions affecting the vagus nerves between the nucleus ambiguus and the ganglion nodosum. Surgical treatment of this condition would require division of either the muscle itself, or its sympathetic nerve supply.

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A DEVICE FOR INTRA-ESOPHAGEAL AND OTHER INTRA-LUMEN PRESSURE RECORDING.

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In a study of the swallowing mechanism described elsewhere,[1] the need for a pressure sensitive capsule arose. Pressure changes within the esophagus produced by contraction and relaxation of the cricopharyngeus muscle were to be recorded. These pressures are difficult to record, as the cricopharyngeus moves up and down during the act of swallowing, changing the relationship of the muscle to any stationary device in the esophageal lumen.[1] A balloon connected by air-filled tubing to an electronic recording system had not produced successful results. Brody and Quigley have called attention to the unsatisfactory physical characteristics of balloons. Constriction of the balloon produces intraballoon pressures which do not accurately reflect pressures within the space being studied,[2] and in the study of the swallowing mechanism[1] the pressure changes recorded by the intra-esophageal balloon could not be consistently reproduced. To overcome this difficulty it was decided to use a pressure sensitive capsule of cylindrical shape and of such dimensions that it would fit within the esophagus at the level of the cricopharyngeus muscle, be in contact with the muscle at all times, yet permit the muscle to slide over it when swallowing occurred. It was important that the entire surface of the capsule be capable of responding to alteration in pressure, and that the effect of this response be transmitted to a system which would make a permanent visual record of the pressure variation. As a qualitative record of pressure change fulfilled the needs of the experiment, exact quantitative estimate of pressure was not attempted except as required for calibration of the system.

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The pressure sensitive capsule described below (see Fig. 1), a modification of the principle described by Lilly and Buchtal and Warburg[4] met the desired requirements. A miniature differential transformer of the type described by Gauer and Gienapp[5] was considered, but discarded because it records only end pressures. Here the sensitive structure is essentially a variable condenser, the capacitance of which varies when

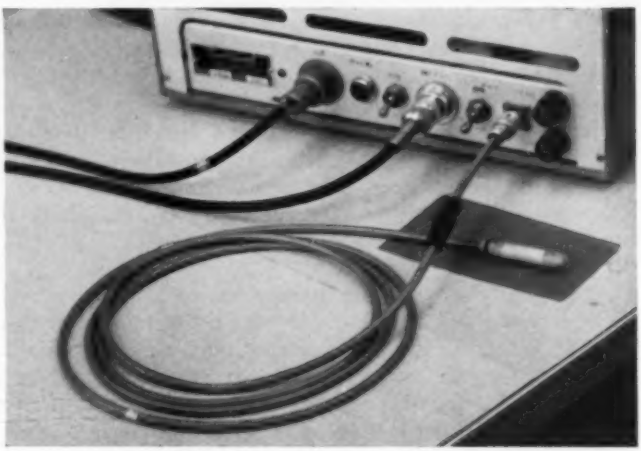

Fig. 1. Pressure sensitive capsule, cable and coil attached to the oscillator-amplifier.

the two metallic surfaces separated by air come closer to each other, or move farther apart, as one is subjected to greater or less pressure than the other. Aluminum foil was used for the metallic surfaces, but instead of air a thin sheet of rubber which forms a solid, flexible dielectric was employed to keep them apart. The layers of foil and rubber were arranged concentrically to form a cylinder 1 cm. in diameter and 5 cm. long. In order to record directly in a body cavity, rather than remotely as part of a gas or fluid filled system, a flexible water-tight cover protected the condenser from conducting solutions such as fluids in the esophagus, which could make a short circuit between the metallic surfaces.

The capsule was mounted at one end of a coaxial cable,* 0.5 cm. in diameter and 266 cm. long, to permit insertion in the body cavity as well as attachment to an oscillator-amplifier. The coaxial cable is an important part of the arrangement, as it consists of a central wire separated from a braided shield in such a way that the spatial relationship of these two remains constant, and the capacitance of the cable varies as little as possible. Since cable and pressure capsule are part of the same electrical circuit, any variation in cable capacitance would simulate a change in capacitance of the pressure capsule and give a false indication of pressure change. The outer braided wire of the cable is attached to the grounded side of the electrical circuit, and, therefore, shields the inner wire from unwanted induced currents in the environment. Advantage is taken of this shielding effect when connecting the pressure capsule to the cable, as the outer of the two metallic surfaces of the condenser is attached to the outer braided wire of the cable.

In order to function properly the pressure sensitive capsule must be in series with a coil of just the right number of turns of No. 34 wire, so that together coil and variable condenser constitute a resonant circuit tuned to the oscillator or carrier frequency of the oscillator-amplifier system to which the cable is attached. A unique feature of the resonant circuit, as used in this case, is the separation of coil and pressure capsule. The coil is inserted in the cable at the end opposite to the pressure capsule, 9 cm. from the connection to the oscillator-amplifier. This avoids placing the coil in a body cavity where it would be exposed to temperature and pressure changes that alter its inductance. Such changes in inductance would change the resonance of the circuit independently of the variable condenser, and produce unwanted deflections on the record not necessarily related to events occurring at the capsule end.

The oscillator-amplifier used† is similar in design to that described by Tomkins.[5] When the resonant circuit consisting of pressure capsule, coaxial cable and coil are attached to the input of this amplifier, they form one arm of an alternating

*Belden No. 8447.
†Manufactured by Technitrol Engineering Co., Philadelphia, Pa.

current bridge which is balanced resistively but slightly unbalanced reactively. When the capacitance of the condenser varies, the reactive unbalance varies and the transmission of the bridge increases. The output voltage of the bridge is proportional to the reactive unbalance, and is changed in the amplifier circuit from an amplified radio-frequency signal to direct current. The pulsating direct current, an image of pressure change at the capsule is then used to drive the re-

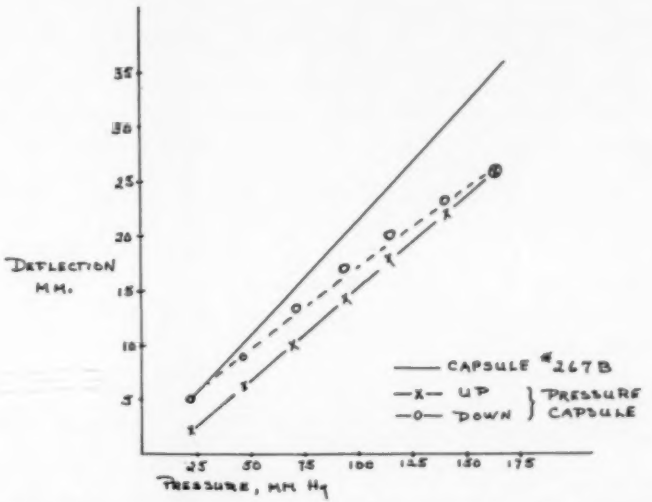

Fig. 2. Response to pressure change of the capsule described, compared to Sanborn capsule No. 267B.

corder, which in this case was a Sanborn Viso-Cardiette direct writer.

To determine whether it would follow pressure changes faithfully enough for the purposes of the experiment, a comparison of the performance of the capsule described was made with the Sanborn capsule No. 267B. This was accomplished by placing both capsules in the same air-filled system. The output of the oscillator-amplifier was fed into a DC coupling amplifier, which was one channel of a four channel recorder. The

Sanborn capsule was attached through its preamplifier to another channel of the same recorder. The response of the Sanborn capsule was known to be linear, and the pressure of the system was varied in increments of 25 mm. Hg. as measured by the calibration of the Sanborn capsule. The deflection in mm. of the two capsules has been plotted against pressure, as shown in Fig. 2, which indicated a fair linear response. A good response to quick changes in pressure was observed when the pressure in the system was alternated two-and-a-half times a second.

It is probable that the capsule could be made smaller, because of the inherent sensitivity of a radio-frequency device. It may also be possible by further refinement in construction to increase the linearity of its response. In addition to the application for which it was designed, there are other situations where it is desired to measure changes in intralumen pressure, and a transducer might have advantages. Brody and Quigley compare various methods for recording these, and discusses their experience with them.[2] One application which could be of considerable value, would be a direct recording of the response of bronchial musculature to various drugs.

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INTERMITTENT PAROTID SWELLING DUE TO ILL-FITTING DENTURES—AN ENTITY; ITS DIAGNOSIS AND TREATMENT.*†

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Renewed interest in sialography, with significant improvement in its technique during the past few years, has provoked many studies of salivary gland disease and a number of original observations. Notable among these has been a better understanding of the etiology, diagnosis, and treatment of inflammatory conditions of the major salivary glands. Sialography, the process of demonstrating roentgenographically the salivary duct system after the injection of radiopaque material, has given us valuable aid in the diagnosis and classification of obstructive and non-obstructive inflammatory lesions.[1,2] Secretory sialography[3] clearly indicates that both conditions are responsible for characteristic changes in the anatomical configuration and physiological function of the major salivary glands.

We have become particularly interested in one such obstructive phenomenon and its mode of management; namely, obstructive sialodochitis. We believe it to be an entity due to an inflammatory stricture at the papilla of the duct developing as the result of trauma from improperly fitting partial or complete dentures. This condition appears to have a natural history, recognizable clinical manifestations, diag-

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Fig. 1, Case 1. Unilateral inflammatory cyst of the parotid gland.

nostic radiographic features, and definite histopathologic changes.

Until recently the management of this condition has consisted of frequent duct dilatations and the administration of antibiotics and chemotherapeutic agents for the relief of swelling and pain incident to the inflammatory reaction with-

in the gland. We should like, therefore, to describe the diagnostic features and pathogenesis of this condition, and to offer a simple operative procedure for its alleviation.

PRESENTATION OF CASES.

Case 1. G. A., No. 781775, a 72-year-old widow, was admitted to University Hospital on April 22, 1954, for evaluation and treatment of a left parotid swelling of eight months' duration; the enlargement was noticed for the first time after eating. There was pain, characterized by "a festering ache and a ropy feeling" in the left cheek. Over a four-week interval the swelling did not recede completely, but fluctuated at meal times between the size of a "kernel and large lima bean." The size of the parotid mass remained stationary for three months prior to admission.

The patient had been wearing complete dentures for more than 30 years. Six years prior to admission, she noticed that the upper dentures became increasingly mobile and often embarrassed her by falling upon the lower lip when opening the mouth; this was also troublesome during mastication. Various "cements" were used in an effort to hold the upper plate in position.

Physical examination revealed a 3x4 cm. mass in the posteromedial portion of the left parotid gland which transmitted light (see Fig. 1). The remainder of the parotid gland was diffusely palpable but not nodular. When the mouth was opened the upper denture fell on the lower lip. There was considerable front to rear and side to side movement of the upper denture. The parotid papilla on each side was hypertrophic and rigid. Stensen's duct orifice was patent, and the duct, which was cord-like, was palpable throughout its buccal course. Pressure over the gland gave rise to a profuse flow of clear saliva, without an immediate return of parotid swelling. Lemon stimulation produced a similar condition. A size No. 1 olive-tipped probe inserted into the duct revealed a fusiform stenosis 0.5 cm. from the orifice. At this point the wall of the duct imparted a gritty sensation. There were no additional strictures in the bucco-masseteric course of Stensen's duct. On the contrary, the duct felt diffusely dilated. Further dilation of the duct orifice preparatory to sialography promoted an active flow of saliva with lemon stimulation.

A smear of the saliva collected by polyethylene catheterization failed to reveal any cells, organisms or crystalline material. The culture yielded organisms of the non-pathogenic actinomyces species which were considered contaminants. The total volume of parotid saliva secreted by continuous lemon stimulation after overnight fasting and collected by polyethylene catheterization was 16.4 c.c. in 60 minutes (see Table I).

Films of the left parotid sialogram taken after injection of 1.0 c.c. of Pantopaque showed elongation, tortuosity and dilation of Stensen's duct without evidence of stricture in its bucco-masseteric course (see Fig. 2). This was accompanied by displacement of the secondary and tertiary ducts about a space-occupying lesion within the left parotid gland. There was minimal retention of contrast material on the post-evacuation films. This sialographic picture was compatible with an inflammatory cyst and chronic main duct obstruction. Aspiration of 17 c.c. of fluid was followed by injection of an equivalent amount of 30 per cent Urokon to outline the cyst cavity (see Fig. 2).

A subtotal parotidectomy was performed with excision of the cyst.

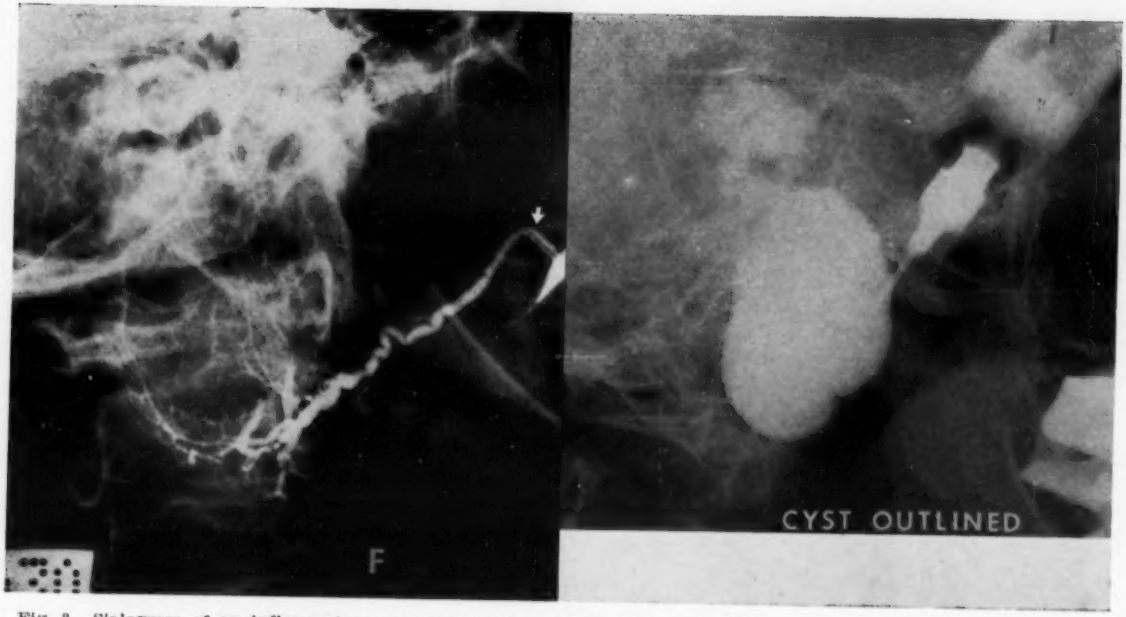

Fig. 2. Sialogram of an inflammatory cyst associated with main duct obstruction.
The Filling Phase shows elongation, tortuosity and dilation of Stensen's duct with evidence of stricture at the orifice only. The stricture has been widely dilated at the arrow. Aspiration of 17 c.c. of fluid, followed by injection of ar equivalent amount of 30 per cent Urokon to outline the cavity of the cyst.

There was considerable scar tissue in the wall of the cyst with evidence of chronic inflammation in the salivary gland (see Fig. 3).

Case 2. K. S., No. 875192, a 58-year-old housewife was admitted to University Hospital on July 9, 1957, with painful bilateral swelling of the parotid gland. The patient was well until April, 1942, when suddenly she noted painful right parotid swelling while eating. A diagnosis of mumps was made by the family physician. Subsequent episodes occurred approximately every three months, all associated with eating. In October, 1946, the left parotid gland was similarly involved.

In 1938 upper and lower complete dentures had been made for the patient, but they did not seem to fit satisfactorily. During the ensuing

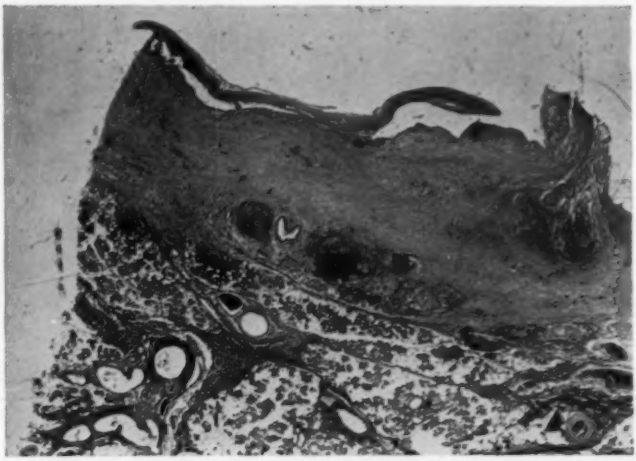

Fig. 3, Case 1. The microscopic section shows considerable scar tissue in the wall of the cyst with chronic inflammation.

years, other dentures were provided, but they never were entirely satisfactory for mastication.

Physical examination revealed bilateral diffuse parotid enlargement (see Fig. 4). The upper denture fell upon the lower lip when the mouth was opened. The lower denture had considerable anteroposterior mobility, and was observed to override each caruncula salivaris at the site where the submaxillary duct (Wharton's duct) opens into the floor of the mouth. Both parotid papillae were hypertrophic and scarred. The salivary flow from each parotid gland was sluggish. Upon stimulation of salivary secretions by sucking a lemon slice, the face swelled gradually, and the flow of saliva into the mouth came in spurts. Probing of each Stensen's duct revealed a stricture at the duct orifices, as well as additional strictures along the bucco-masseteric course of Stensen's duct. The wall of each Stensen's duct imparted a gritty sensation upon probing. There were no strictures in either submaxillary (Wharton's) duct.

A smear of parotid saliva collected by polyethylene catheterization revealed desquamated cuboidal epithelial cells. No organisms were observed by smear or culture from either side. The submaxillary saliva showed no cells or organisms. Total volume of parotid saliva secreted by continuous lemon stimulation after overnight fasting was 17.7 c.c. in 60 minutes for the right parotid gland and 18.1 c.c. in 60 minutes for the left (see Table I). A quadriglandular sialogram was performed. An injection of 0.9 c.c. of Pantopaque in each Stensen's duct revealed a mark-

Fig. 4, Case 2. Photographs taken before and one week after bilateral parotid dochoplasty.

edly dilated and irregularly segmented main duct. The submaxillary ducts (0.35 c.c. Pantopaque injected in each duct) were similarly involved, although the degree of dilation was not so pronounced as in the case of Stensen's ducts (see Fig. 5).

On July 16, 1957, a bilateral dochoplasty of Stensen's duct was performed.

Case 3. M. B., No. 839162. A 46-year-old wife of a physician came to University Hospital in April, 1956, because of repeated painful swellings of the left parotid gland. This patient had been asymptomatic until May, 1955, when she noted a sudden post-prandial left parotid swelling. This

was associated with a low-grade fever. Subsequently, she complained of a feeling of tightness and dull pain in the bucco-masseteric region while eating. The diagnosis of mumps was made at the time of the original attack. During the next ten months there were similar episodes of pain and swelling in the left parotid area approximately once a month, and invariably associated with her meals. The swelling receded spontaneously on each occasion.

Upper and lower partial dentures had been made for this patient in 1944 for the purpose of correcting her bite. She stated that the upper dentures became loose frequently and had to be "tightened." She preferred to chew her food on the right side.

Fig. 5. Sialogram of advanced main duct obstruction with secondary infection. The Filling Phase reveals that each Stensen's duct is markedly dilated and irregularly segmented.

Physical examination revealed a moderately swollen, diffusely palpable left parotid gland. The left parotid papilla was hypertrophic. A scanty amount of clear gelatinous saliva could be milked from the duct orifice. Upon stimulation by sucking a lemon slice, the left parotid gland swelled and a firm ropy Stensen's duct could be palpated along its bucco-masseteric course. Upon further milking of the duct system by massage, saliva entered the mouth with a squirt. Probing revealed a stricture 0.5 cm. proximal to the duct orifice, and only a size No. 1 olive-tipped probe could be passed beyond the stricture. Additional strictures were also encountered along the bucco-masseteric course of Stensen's duct. The wall of the duct imparted a gritty sensation upon probing. The saliva contained desquamated cuboidal cells, but no organisms were found in the smear or culture. The total volume of parotid saliva secreted by continuous lemon stimulation (after overnight fasting) was 20.5 c.c. in 1 hr. on the left side, and 20.0 c.c. in 1 hr. on the right (see Table I). This secretion was obtained after the stricture near the orifice had been widely dilated.

The sialogram revealed multiple strictures of Stensen's duct on the left side after injection of 0.8 c.c. Pantopaque. There was virtually complete retention of the contrast material on the five-minute post-evacuation film (see Fig. 6). The filling and excretory films of the right parotid gland and submaxillary glands were normal.

On June 15, 1956, a dochoplasty was performed.

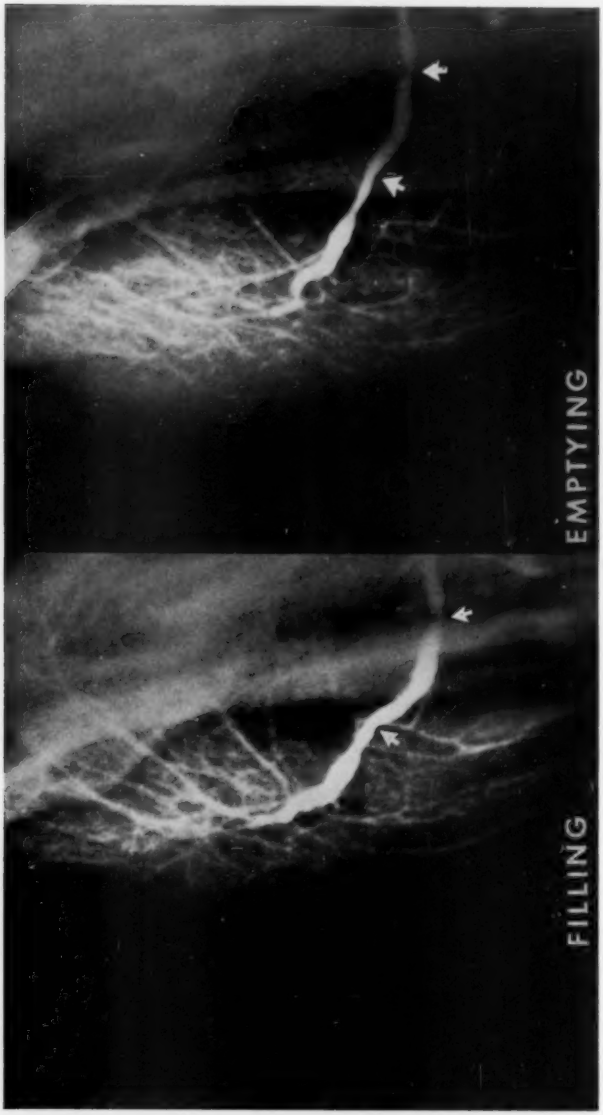

Fig. 6. Sialogram of uniform dilation of Stensen's duct with beading (2X enlarged). This appears to be an early anatomic alteration characterized by stricture formation (arrows) at the duct orifice and along the bucco-masseteric course of Stensen's duct. In the 5-Minute Post-Evacuation Film, the obstruction to salivary flow is verified by the unaltered appearance of the sialogram.

THE CLINICAL PICTURE.

As noted by other investigators,[4,5] the condition is associated with partial or complete dentures which are ill-fitting. Diffuse parotid swelling with pain and a feeling of pressure in the cheek frequently occurs while eating. When salivary flow is stimulated by a potent sialogogue (lemon) the gland may swell. Upon massage of the parotid region, only a slug-

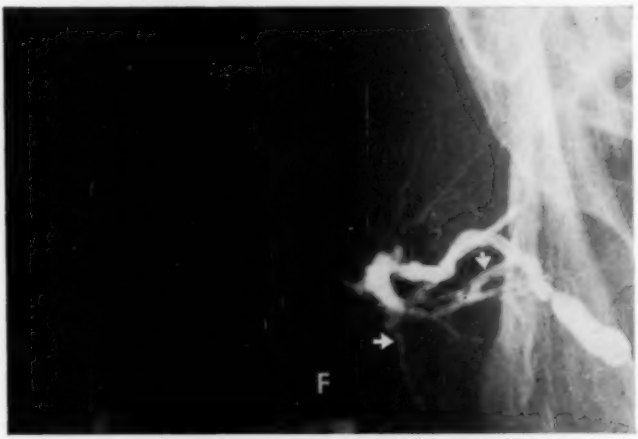

Fig. 7, Case 2. A sialogram of the Filling Phase (2X enlarged) demonstrates that the secondary ducts (arrows) may also be involved in obstructive sialodochitis; however, there is no alteration of the most peripheral radicals. This is the differential point between obstructive sialodochitis and non-obstructive sialectasis.

gish flow of clear saliva from a hypertrophic parotid papilla is noted. The cord-like dilated Stensen's duct can usually be palpated along its bucco-masseteric course. Upon probing the duct, dilated segments alternate with strictures. A gritty sensation imparted to the probe by the hypertrophic and metaplastic duct epithelium (see Fig. 9) may be mistaken for calcareous material.

THE PATHOGENESIS.

This condition appears definitely to be associated with the

Fig. 8. Case 2. The Filling Phase of this quadriglandular sialogram demonstrates the markedly dilated and irregularly segmented main ducts (Stensen's ducts). The submaxillary ducts (Wharton's ducts) can be similarly involved, though the degree of dilation is not so pronounced. There is no alteration of the peripheral duct architecture. The Post-Evacuation Film indicates that the obstruction at the outlet (orifice) is the principal deterrent to evacuation.

FIG. 9, Case 2. Microscopic sections taken from, (A) the constricted orifice of Stensen's duct; (B) the greatly dilated portion of the main duct just proximal to the stricture. There is metaplasia, scar tissue formation and stenosis at the site of the stricture. This is consequent upon traumatisation of Stensen's duct orifice in the parotid papilla.

presence of dentures. The patient is required to alter the mechanism of mastication in a manner which does not sustain the normal chewing habit. As food is being chewed on one side of the mouth, the parotid papilla on the opposite side tends to be sucked mesially and bitten. Mastication is further rearranged and altered when alveolar absorption occurs, allowing the dentures to shift about while chewing.

When the parotid papilla is subjected to repeated trauma and inflammation, fibrous tissue proliferation and scar tissue cicatrization occur. This interferes with normal salivary flow, and the ensuing pressure behind the stricture results in dilatation of the gland duct. It is not long before secondary bacterial infection supervenes with a diffuse inflammatory reaction along the bucco-masseteric course of Stensen's duct and with production of multiple inflammatory strictures. If a stricture in the secondary or tertiary ducts seals off a section of secreting acinar tissue an inflammatory cyst may be formed (see Fig. 2).

THE SIALOGRAM WITH CLINICAL AND HISTOLOGICAL CORRELATION.

The diagnostic features of this condition are dilatation and stricture formation in the main salivary duct *without the terminal peripheral duct involvement* seen in sialectasis. The degree of anatomic alteration depends on the frequency, severity and duration of: *1.* Obstruction caused by injury to the parotid papilla; *2.* Secondary bacterial infection associated with interference of salivary flow.

Among the patients above described, three roentgenographic patterns can be distinguished.

1. ELONGATION, TORTUOSITY AND DILATATION OF THE MAIN DUCT.

These changes are noted when a stricture is present near the duct orifice (see Fig. 2). Filling phase films and probing demonstrated the stricture. Some of the secondary and tertiary radicals share in the dilatation. If a stricture in the minor ducts resulting from a parotid infection seals off a

section of secreting acini an inflammatory cyst may be formed. A monolocular cyst with considerable scar tissue in its wall and with surrounding parenchymal pressure atrophy is seen in Fig. 3. The stricture was probably the result of a parotitis eight months prior to admission to the hospital, the only such episode experienced by the patient.

2. UNIFORM DILATATION OF STENSEN'S DUCT WITH BEADING.

This appears to be an early anatomic alteration of the duct characterized by stricture formation at its orifice and along its bucco-masseteric course. We note these changes in Stensen's duct of patient (Case 3). The emptying film shows virtually complete retention of the contrast material (see Fig. 6). The mechanism of retention is no doubt related to the checking action of the salivary flow by multiple strictures along the course of the duct and the non-distensible stricture at the duct orifice.

3. MARKED DILATATION AND IRREGULAR SEGMENTATION OF THE MAIN DUCT.

These changes are seen in patient (Case 2), in whom ill-fitting dentures were worn for 19 years, with recurrent episodes of parotid swelling. As noted in the filling phase, the secondary ducts are also involved, but the most peripheral radicals do not participate in the inflammatory process (see Fig. 7). This represents an advanced composite picture of main duct obstruction and secondary bacterial infection. The submaxillary ducts can be similarly involved if the lower denture is faulty, although the degree of dilatation is not pronounced. The post-evacuation film demonstrates that the obstruction at the outlet is the principal deterrent to evacuation (see Fig. 8). When the stenotic duct orifices are removed surgically and a duct-plasty is performed, the continuous flow of saliva is re-established and the parotid swelling recedes. Histologic sections of the constricted duct orifice show metaplasia, scar formation and stenosis that are the result of trauma of Stensen's duct in the parotid papilla (see Fig. 9).

DISCUSSION.

Obstructive sialodochitis due to ill-fitting dentures is not a common disorder when compared with salivary gland inflammation caused by calculi or by the non-obstructive sialodochiectasis (sialectasis) group of diseases such as recurrent pyogenic parotitis, Sjögren's syndrome and Mikulicz disease. Only three such cases (2.7 per cent) have been observed in a total of 138 patients referred to the clinic for various inflammatory lesions of the salivary glands since July, 1953. These cases also represent only 0.75 per cent of 352 sialograms made since the above date. Because of the morphologic pattern of main duct "estasia" produced by alternating dilatations and strictures, the condition belongs to the obstructive sialodochiectasis group as classified by various investigators[1,4,7]; however, obstructive sialodochitis should not be confused with non-obstructive sialectasis. In the former, there is no alteration in the terminal ducts nor diminuation in the volume of saliva secreted.[5] In the latter, however, we note dilatation of many of the terminal ducts, and a definite diminution in the quantity of saliva secreted by the diseased gland.

TABLE I.—SECRETION VOLUME STUDIES OF PURE PAROTID SALIVA IN C.C./HR.

Method of Collection: Via Polyethylene Catheter.
Stimulation: Feeding Continuous Lemon Slices.

Normal Female, Age 44: 21 c.c.	Case 3.	Age 46:	20.5 c.c. Left Parotid; 20.0 c.c. Right Parotid.
Normal Female, Age 60: 17.5 c.c.	Case 2.	Age 58:	17.7 c.c. Right Parotid; 18.1 c.c. Left Parotid.
Normal Female, Age 70: 17.0 c.c.	Case 1.	Age 72:	16.4 c.c. Left Parotid.

In obstructive sialodochitis, associated with ill-fitting dentures, the volume of pure parotid saliva secreted in c.c. per hr. does not appear to vary from the normal (see Table I), and this probably explains the normal architectural pattern of the terminal ducts seen roentgenographically. Possibly, too, it accounts for the fact that no pyogens or inflammatory cells are present. Thus, the mechanism which resists retrograde infection of salivary parenchyma by mouth organisms[6] can operate successfully in the face of increased pressure gradients

of dilatation and strictures, as long as the normal complement of acinar tissue is available and sufficiently productive to wash the organisms out of the duct.

Anatomically, two types of duct obstruction are apparently produced—that which is noted at the duct orifice and obstruction along the bucco-masseteric course of the duct. The

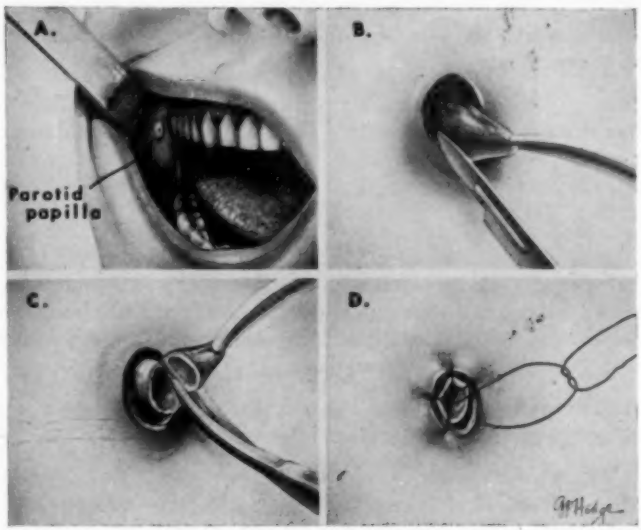

Fig. 10. Technique of Dochoplasty. A.—Parotid papilla opposite second molar tooth. B.—The parotid papilla is grasped and the stenotic portion of Stenson's duct is dissected free from the surrounding buccal tissue. C.—The papilla is amputated. D.—The open end of the duct is sutured with fine silk to the surrounding mucous membrane.

duct orifice obstruction is caused by trauma to the parotid papilla. The bucco-masseteric obstruction appears to be a more advanced stage of the condition resulting from secondary basterial infection and inflammatory reaction along the bucco-masseteric course of Stensen's duct.

TREATMENT.

The treatment is surgical. The technique of dochoplasty is

simple enough. Under local anesthesia (procaine 1 per cent) injected about the orifice of Stensen's duct, the papilla is grasped and amputated (see Fig. 10). This simple procedure exposes the stenotic duct, which is then dissected free from the surrounding tissue for a distance usually of something less than a centimeter until its dilated portion is identified. At this point the duct stands out conspicuously because of its marked dilation. The open end of the duct is then sutured, with fine silk, to the surrounding mucous membrane in a manner which exposes the duct to free communication with the oral cavity.

Antibiotics and chemotherapeutic agents may be used for several days postoperatively. Healing is kindly and prompt. Rapid regression of parotid swelling occurred in the three patients herein reported, and they have remained asymptomatic to date.

SUMMARY.

1. Ill-fitting dentures can cause the syndrome of obstructive sialodochitis with intermittent parotid swelling. We believe it can be regarded a definite entity.

2. The pathogenesis and clinical picture are presented with radiographic and histologic correlation.

3. Secretory sialography is useful in making the diagnosis and in differentiating it from non-obstructive sialectasis.

The histologic picture is that of stenosis, scar tissue formation, and metaplasia of the duct epithelium; all compatible with traumatization of the parotid papilla.

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GRANT TO JEWISH HOSPITAL, ST. LOUIS.

The receipt of a $5,000 grant from the Louis D. Beaumont Foundation for the furtherance of research in the field of surgical rehabilitation of hearing at the Jewish Hospital of Saint Louis was announced recently by Dr. Ben H. Senturia, Director of the Department of Otolaryngology. The check was presented to the Hospital by Mr. Morton J. May, Trustee of the Foundation.

The surgical rehabilitation program is a recently expanded one, functioning on both clinical and research levels. It includes comprehensive pre- and post-operative hearing testing; an evaluation of the function of the Eustachian tube, and surgical correction of the pathologic changes which involve the ear drum, ossicular chain and middle ear.

EFFECT OF HORMONES ON HYPOGAMMAGLOBULINEMIA.*†‡

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A deficiency of gammaglobulin is found in certain cases of chronic upper respiratory infection. Often, this type of problem case will improve while being given the appropriate antibiotic, only to regress into the chronic ailment when such medication is stopped. It has been known that Gammaglobulin, given parenterally, will give protection for periods of three to four weeks. This is a report of studies to see whether thyroid and steroid hormones can bring the Gammaglobulin back to more normal levels with relief of the infection.

Recently, Good[1] studied "Experiments of Nature" to see whether an understanding of the mechanisms of antibody and Gammaglobulin production can be found by studying the characteristics and adjustment of tissues in patients suffering from extreme disturbances of the immunological mechanism.

His work centered chiefly on agammaglobulinemia which he feels has four cardinal features: *1.* Increased susceptibility to infection; *2.* Absence of Gammaglobulin from the blood and tissues; *3.* Absence of circulating antibody from the blood and tissues; *4.* Failure of antibody production in response to antigenic stimulation.

He recognizes three forms of this disease: *First*, Bruton's,[2]

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a congenital disease in which the basic difficulty is an inborn error of metabolism transmitted as a sex-linked recessive trait demonstrable clinically only in males; *second*, an acquired form of agammaglobulinemia which occurs in either sex and has its onset at any age; *third*, an extension of the neonatal agammaglobulinemia beyond the time at which normal production ordinarily begins. In these cases, he finds a generalized absence of lymphoid tissue including tonsils and adenoids, with the lives of the patients a constant succession of bacterial infection, most frequently encountered as chronic lung disease, sinusitis, otitis media, pharyngitis and occasionally either a sprue-like syndrome or recurrent bacterial meningitis.

It occurred to us that in studying clinical patients, instead of studying the agammaglobulinemic, much could be learned from the patient with hypogammaglobulinemia. Electrophoretic patterns were performed on 126 patients with chronic recurrent infections, which would respond temporarily to antibiotics but would recur when they were stopped. In an effort to keep the results practical, the tests were made at easily available commercial laboratories rather than by our own highly specialized technicians; for while this might diminish the accuracy slightly, it would provide a tool available to most physicians. The results fell into three main categories: the first, consisted of 58 patients who had normal Gammaglobulin levels; the second group of 24 patients, chiefly Navajo Indians, in which there was a marked increase in Gammaglobulin; and the third group of 44 people in which there was a deficiency. This is a study of these cases which had at least a 40 to 50 per cent deficiency.

Twenty-one patients of ours fall into the deficiency classification. Five have been reported previously.[3] Of the 16 subsequently found, 13 suffered with chronic pharyngitis, having large, raised plaques of lymphoid tissue in the pharynx and nasopharynx, and slight to moderate glandular adenopathy of the cervical chain. Of the remaining, two patients had chronic draining ears, and the last, a sinus infection.

One of the ear problems had been subjected to a mastoidec-

tomy twice, once in the Service 14 years ago, and a second radical operation three years ago. Despite the surgery, the ear continued to drain and granulations kept forming very rapidly, resisting all attempts to remove them. One antibiotic after another was used without benefit until the organism (Pseudomonas Aeruginosa) developed resistance. An electrophoretic pattern of serum proteins demonstrated the suspected deficiency. He was given 10 cc. of Gammaglobulin intramuscularly every four weeks, the appropriate dosage for his weight. This treatment has continued for a period of nine months. The ear has dried during this time, and the granulations are slowly disappearing. He is being treated with the appropriate antibiotic in addition to this therapy, but while there were no results on the antibiotic alone, the Gammaglobulin plus the antibiotic has so far been quite successful. It is interesting to note that electrophoretic studies still show the same deficiency present as existed when the therapy was started, despite the clinical improvement.

As reported a year ago,[3] it was found that when the antibodies were not being adequately secreted, steroids might allow the cells, lymphocytes and/or plasma cells to "secrete" retained Gammaglobulin.

Of the five cases reported, one has not returned. Three are well clinically after receiving Meticortelone, 25 mgms. intramuscularly every three weeks for several months. They now return only periodically for check-up. The fifth is still under treatment, and still reports for 25 mgms. of prednisolone intramuscularly every month. As long as he has this he is free of sore throat, but on the three occasions when he has omitted the treatment, the pharyngitis returned, though less severely. The electrophoretic pattern still shows a definite Gammaglobulin loss; but it is slowly returning to normal, and the lymphoid hyperplasia is clearing (see Fig. 1).

It occurred to us that since thyroid produced an increased lymphocytosis[4] followed by a later parallel mononuclear production, and increased protein metabolism,[5] it might increase the circulating Gammaglobulin. It also was possible that small doses of thyroid, which would enhance steroid production via

the pituitary, might also increase Gammaglobulin production. It was realized that profound changes in resistance are not necessarily associated with either hypo- or hyper-thyroidism, but dysfunction of the thyroid has been related to resistance through the above mentioned mechanisms. Since the thyroxine has been reported to increase opsonin production,[6,7]

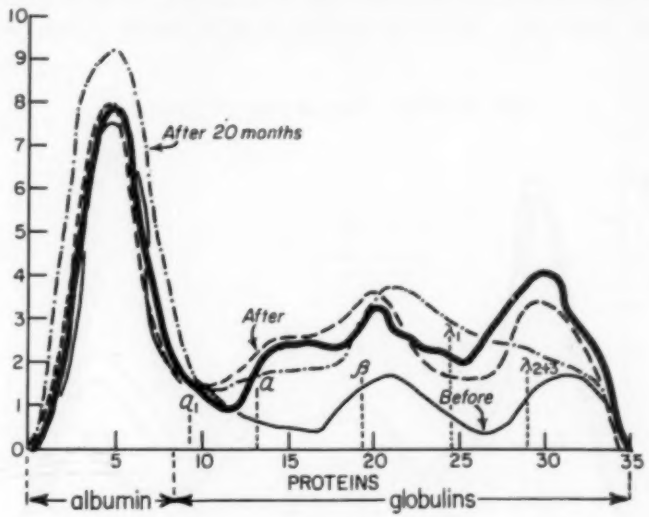

Fig. 1.

it was felt that an iodine-containing portion of thyroid secretion would be the most desirable to use.

Sodium liothyronine (Cytomel*) was thought to be the most desirable iodine containing thyroid extract for the study. Its action[8] is believed to be directly on the peripheral tissues, and immediate results could be expected without awaiting intermediate metabolic conversions. It is effective in some cases where other thyroid hormones fail. In addition, because of its rapidity of onset of action, symptoms of overdose would be recognizable promptly enough for safe with-

*The Cytomel was generously supplied by Mr. John Simon, of Smith, Kline & French Laboratories, Philadelphia, Pa.

drawal. It is absorbed readily from the gastro-intestinal tract, and rapidly cleared from the blood stream, being more loosely bound to the plasma proteins than thyroxin; hence, the oral administration would be the most satisfactory as well as the most effective.

The first patient in whom the use of this drug was attempted was a five-year-old child with chronic Proteus middle ear infection. Here, one antibiotic after another had been

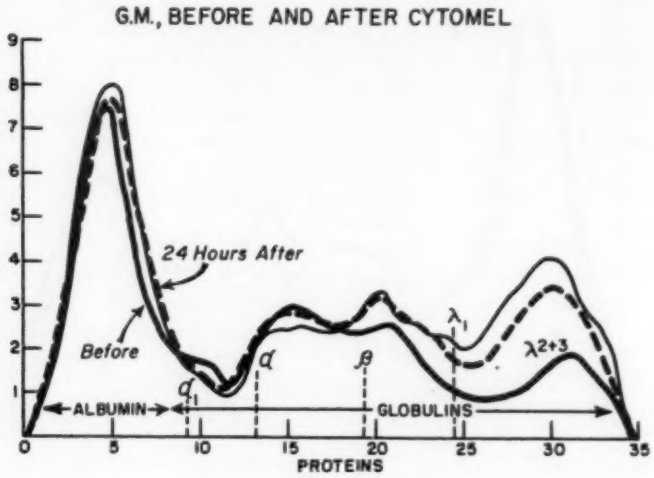

Fig. 2.

used until they had lost their capacity to destroy the infection. After each subsequent cold, there was a return of the running ear, until no further medication was available. Electrophoresis at this time confirmed a low serum Gammaglobulin level (see Fig. 2). He was given five micrograms of Cytomel a day for four days without any effect; however, when the dosage was raised to ten micrograms of the sodium liothyronine a day for four days, the Gammaglobulin was raised to almost normal levels, and the ear improved tremendously. This clinical effect lasted for about three weeks.

Since that time he has been given one dose of ten micrograms of Cytomel for four days, every three weeks. As long as this is taken, his ear remains dry. With colds, he is given an appropriate prophylactic antibiotic. Although it is realized that this will not affect the Proteus, it is believed this will keep down the coccal forms which may allow the Proteus to

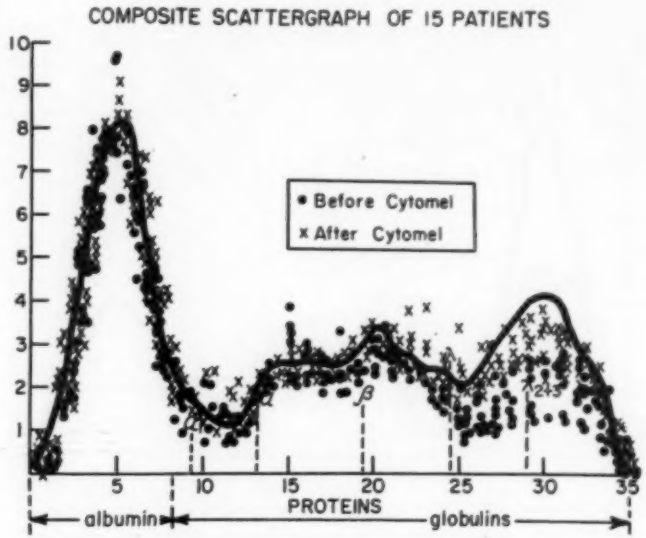

Fig. 3.

start spreading. With this combination his infection has been kept under control for the past eight months.

Fifteen patients with chronic pharyngitis and hypogammaglobulinemia followed the same pattern. Thirteen of these were adults and two children; one a child of one of the adults. The adults were maintained on ten micrograms of Cytomel for four days. This raised their levels of Gammaglobulin almost to normal (see Fig. 3). This increased level persisted for about three weeks and was coincident with the disappearance of their clinical symptoms. During this period, the

general lassitude, fatigue and throat soreness disappeared. After several courses of the liothyronine the lymphoid nodules began to diminish in size as well as redness. The electrophoretic patterns taken at the end of a three-week period usually demonstrated that the levels had diminished to the low present at the onset of treatment. The children responded similarly to doses of five micrograms of Cytomel a day for

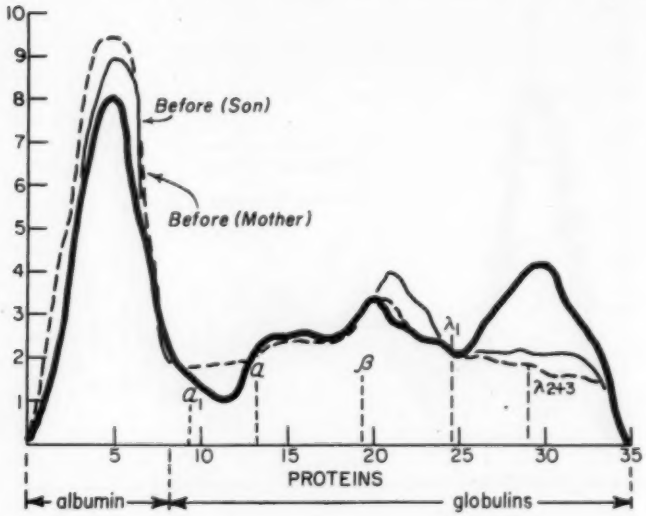

Fig. 4.

four days. It is interesting to note that of these two patients, a mother and son, had demonstrated very similar types of curve (see Fig. 4).

In three cases where 25 micrograms of Cytomel were given, the rise of the Gammaglobulin was less than with ten micrograms (see Fig. 5). It is interesting to speculate why the response is limited to a narrow band, since the Gammaglobulin rise is lessened on either high or low doses. This seems to agree with the clinical finding of a correlation of lessened

resistance with dysthyroidism but no effect with hypo- or hyperthyroidism.

Protein bound iodine studies showed most of the levels within normal limits, usually close to the higher level of normal. Very little rise was found with the small doses of Cytomel given.

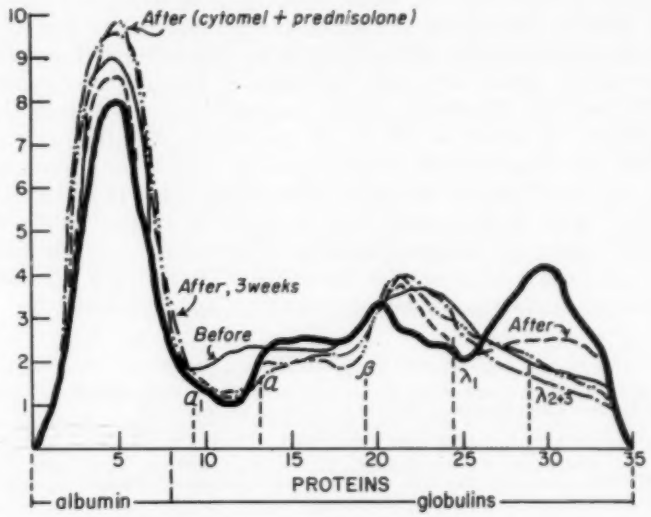

Fig. 5.

Since it is felt that the effect of liothyronine was mediated via the adrenal cortical hormones, an attempt was made to see whether this action could be enhanced by giving the patient 10 to 25 micrograms of Cytomel and five milligrams of prednisolone a day for four days. To our dismay, the combination produced a lesser increase in the Gammaglobulin than did either drug alone, confirming the findings of Summers[9] and Hollender[10] that corticosteroids do not supplement thyroid therapy symptomatically. It is felt that Cytomel may act through pituitary stimulation of the adrenals. When adrenal hormone is also supplied, the stimulus is weakened and hence

the response is poorer. Another possibility is that Cytomel's action is a direct one on the protein fraction transporting the oxidation enzymes.

In summation, further studies confirm our impression of a year ago that there are two types of hypogammaglobulinemia: the most common type is the one reported by Good, in which there is little lymphoid tissue; the second is a numerically much smaller type in which hypertrophied lymphoid tissue is present, but where antibodies are not adequately secreted. In this latter group, administration of liothyronine or cortical steriod hormones stimulates secretion of the lymphoid tissue. The antibody liberated contains sufficient quantity of Gammaglobulin to show an effect on the electrophoretic pattern. This has usually been accompanied by clinical improvement in our small number of cases. This series is not large enough to do more than indicate the necessity of further study to help explain the inter-relationship of hormones and resistance to infection; however, for the clinician, it offers a valuable therapeutic tool in the aid of a small group of patients.

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CAROTID BODY TUMORS.

Report of One Case.*†

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Carotid tumors are not frequent, and few surgeons have had the opportunity of operating on any large number of cases. Small series of cases have been reported by several authors, many with an excellent review of the literature. Morfit and co-authors, in a recent paper, emphasized two points which are brought out in the present case report. First, these tumors are not as benign as previously claimed, and are found to invade locally and even disseminate, providing the cases are followed long enough. Second, it is often possible to remove carotid body tumors without having to sacrifice the large vessels.

This "potato tumor," as it is sometimes called, arises from the carotid body, a small ovoid mass found resting in the loose connective areolar tissue situated on the medial aspect of the carotid bifurcation. Luschka was the first to describe this small organ, composed of an agglomeration of rounded lobules separated one from the other by fibrous septa containing blood vessels. Each of these lobules is nourished by a small arteriole branching from a principal artery derived directly from the common carotid. The cells are closely related to the vessels and are grouped into small nests or strands. The shape of the cell is variable; a spherical nucleus has a fine chromatic network and the cytoplasm is granular, acidophilic and non-chromaffin. Histologically similar non-chromaffin bodies or tissues are present along the aorta, the jugular bulb, the ganglion nodosum, the ciliary body (in the monkey), and finally in the coccygeal body. The carotid is probably the best known of these bodies, forming what is now referred to

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as the "chemoreceptor system." The physiology of this organ is not as yet well understood. Its pathology appears to be limited to tumor formation only.

The presence of a mass or lump in the neck may, for a long time be the one and only symptom of a carotid body tumor. The mass has usually been present and slowly growing for a period of five to six years before the patient consults. In a series of cases reported by Hendrick, the average duration of symptoms was eight years before patients sought relief. The tumor is a slowly growing one. It is deeply situated in the lateral portion of the neck, under the sterno-mastoid muscle, at the level of the carotid bifurcation. The tumor is round or ovoid, firm to palpation and is not fixed to the skin. It is neither painful nor tender. It has been reported as being depressible and as transmitting pulsations. It is movable in a lateral but not in a vertical direction, and does not move on swallowing. As it increases in size the tumor may grow medially and project into the pharynx, or grow up and extend to the base of the skull. Bulging of the pharyngeal wall was found to be a presenting symptom in a good number of cases, and in one reported by Hendrick, was such as to interfere with swallowing. The tumor is usually unilateral, although bilateral cases have been reported. Symptoms due to compression of surrounding structures are next in frequency. Pressure on the recurrent laryngeal nerve, the vagus, the cervical sympathetic, the hypoglossal, the phrenic and the cervical plexus, causing corresponding paresis or paralysis, have all been reported. In the present case the VIIth, VIIIth, IXth, Xth, and XIth cranial nerves were all involved. It is unique in this respect, as no other case was found in the available literature in which so many nerves were affected.

Grossly, a carotid tumor is a firm, reddish-brown, lobulated mass of variable size surrounded by a capsule.

Histologically, Masson states that the tumor very closely reproduces the architecture of a normal carotid body. The glandular lobules separated by fibrous septa containing vessels are increased, not only in number but also in size. This explains the very marked vascularity of these tumors and the

profuse hemorrhage with which their removal is always accompanied. It would appear as a hyperplasia, and for this reason it has once been suggested that these tumors be called "goiters of the carotid body" (Dietrich and Siegmund) (see Figs. 1, 2, 3).

The tumor is encapsulated, with the secondary nodules in turn, also encapsulated. Long strands of cells are separated

Fig. 1. Microphotograph of carotid paraganglioma showing a well defined capsule; X48.

from one another by conjunctival septa, often presenting hyaline degeneration. The cellular nests, or strands, are themselves divided into narrow trabeculae by a very fine conjunctivo-vascular system. The cells are polygonal, with a fine granular cytoplasm and a comparatively small nucleus, which is hyper-chromatic. One or two acidophilic nucleoli are often seen. Two histological patterns are described; the alveolar and the peritheliomatous, in which the cellular units are smaller and more irregular than in the alveolar pattern.

Masson considers most of these tumors benign but at the same time describes a malignant form, which he considers

of low grade malignancy but capable of local and regional invasion. Harrington and co-authors, in their review of 20 cases, reported evidence of malignancy in ten, as shown by active mitotic figures, cellular variations, giant cells and by invasion of the capsule. Their criterion of malignancy is based on histological findings only, and not substantiated by follow-up or post-mortem examination. On the other hand, Pettet and co-authors, after reviewing 314 cases found in the

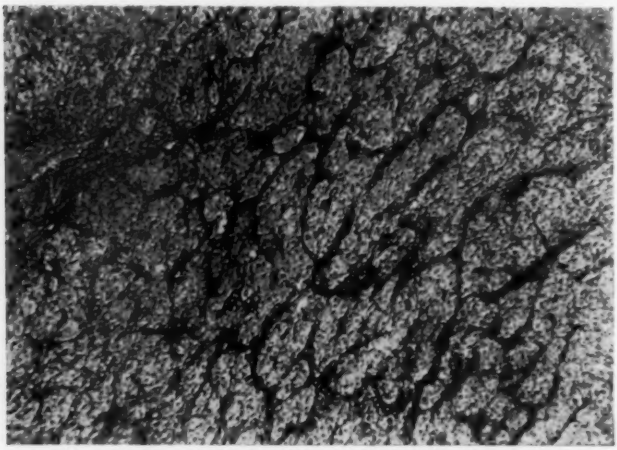

Fig. 2. Cellular strand or nests are seen to be separated from one another by fibrous septa; X60.

literature and 47 cases treated at Mayo, came to the conclusion that these tumors were very rarely malignant. Goormaghtigh and Pattyn report two cases, one of which was malignant with metastases in the cervical, mediastinal and retro-peritoneal nodes, the lungs and lumbar vertebrae, proven at autopsy. It can be concluded that there is no uniformity of opinion in regard to the malignancy of these tumors. It is hoped that with time and a proper follow-up of operated and non-operated cases, a better understanding of carotid body tumors will result.

The diagnosis of a carotid body tumor is not an easy one

to make and few pre-operative diagnoses have been made. The history of a slowly growing, non-tender mass in the lateral portion of the neck, at the level of the carotid bifurcation, movable in a lateral and not in a vertical direction, should make one suspect this type of tumor. Aspiration biopsy has been recommended, but is not without danger, and is very often inconclusive.

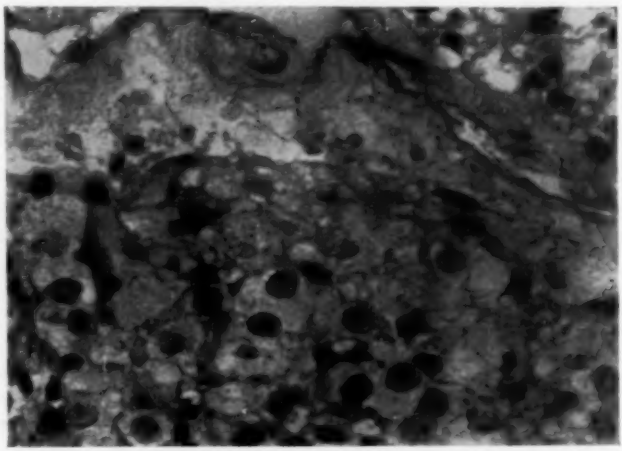

Fig. 3. Higher magnification showing cellular details; the Cytoplasm is granular, acidophilic and non-chromaffin. The nucleus is spherical and presents a fine chromatic network; X418.

Carotid body tumors do not respond to radiation therapy and the treatment remains a surgical one. Until recent years the indication and advisability of surgical removal was often questioned because of the high mortality and morbidity associated with the ablation of these tumors, particularly when excision of the carotid arteries became necessary. It has now been shown that a great number of these tumors can be removed without injury to the vessels, if a careful dissection is done along the serosal plane of the arteries concerned; furthermore, when it becomes necessary to include the carotids along with the specimen, anastomosis or arterial

grafts have been found quite successful in reducing this high mortality rate. This question is well discussed by Conley and others.

A 53-year-old male was referred by the Rehabilitation Center in April, 1954, for a laryngeal examination prior to vocal training, which had been recommended because of a hoarseness of some years standing. The history of this patient's complaints is long and complicated, but most interesting, and I shall briefly summarize it chronologically as follows:

In 1950 the patient first complained of hoarseness and saw a physician, who told him that he had an enlarged right tonsil, for which he recommended some ten treatments of deep radiotherapy. No change in either the hoarseness or the size of the right tonsil resulted.

In 1951 the tonsils were removed, and it was then realized that the right tonsil was not enlarged but was being pushed forward and medially by some extrinsic mass. Histological examination of the tonsil at this time did not reveal any pathology.

Shortly following tonsillectomy the patient noted a small swelling behind the angle of the jaw. According to the patient, this mass has varied from day to day until the present time.

Four or five months later the patient's voice became lower in pitch and less powerful. He also began having difficulty swallowing and reported that he would have frequent choking spells.

In 1952 weakness of the right shoulder and right arm were noted, and wasting of the sterno-mastoid and shoulder muscles quickly set in. At the time of consultation the patient stated that hearing in the right ear had been decreasing for the last six months or so.

X-rays of the chest in 1951 were reported as negative.

Examination (April 21, 1954).

The patient is a well developed and well nourished white

male of about stated age. He talks in a somewhat monotonous and muffled voice which definitely lacks volume. Wasting of the sterno-mastoid and shoulder muscles on the right is quite apparent.

Ears—Canals are normal. Healed perforation in both drums.

Nose—The septum is straight. Mucous membrane is normal in appearance and no secretions are to be seen.

Nasopharynx—There is a smooth bulging of the right lateral nasopharyngeal wall with loss of all landmarks. The mucous membrane covering this mass is normal in appearance.

Oropharynx—Dentures. The tongue is normal in appearance and mobility. The tonsils have been removed and the right tonsillar fossa is seen to bulge forward and medially. This bulging or mass is continuous with that noted in the nasopharynx. The soft palate on the right side and the uvula are deviated to the left. There is no gag reflex.

Larynx—The right cord is paralyzed in abduction; otherwise the larynx is normal. There is considerable pooling of secretions in the right pyriform sinus.

Neck—A marked atrophy of the sterno-mastoid and trapezius muscles on the right side is noted. A firm mass can be palpated behind and below the angle of the jaw. This mass, best palpated by bidigital palpation, is continuous with the mass noted in the oro and nasopharynx. It is firm, non-tender and gives the impression of very slight mobilization in a lateral direction.

A tentative diagnosis of tumor of the neck, probably mixed tumor, with paralysis by pressure of VIIIth, IXth, Xth, and XIth cranial nerves is made. The patient is requested to come to hospital for further investigation and removal of this mass.

The patient was admitted to N.D.H. (4666) on May 2, 1954, and following examinations obtained:

Audiometric—Total loss of hearing on the right.

Vestibular—Complete loss of function of the right.

Blood—RBC, 4,775,000; WBC, 8,250; Hmg., 97. Differential within normal limits.

Urea and blood glucose normal.

Bleeding and clotting times normal.

Urinalysis normal.

Neurological Consultation: (Dr. L. Rinfret) Paralysis due to pressure by cervical tumor of VIIIth, IXth, Xth and XIth cranial nerves. Some fibrillations of the right side of the face are noted, right facial weakness is suggested.

Radiological Examination, May 12. Lungs are normal (C37339). Cranium and cervical region: "Presence of a soft tissue mass bulging into the right side of the nasopharynx, which extends upwards through the base of the skull, into which it has produced a large defect posterior to the petrous pyramid at the level of the jugular foramen; furthermore, there is erosion of the posterior border of the pyramid with complete destruction of the internal auditory meatus. This destruction does not involve the cochlea but has markedly thinned out the petrous bone. This erosion extends posteriorly to the occipital foramen. We are dealing with a large erosion with well defined contours. It has the appearance of a destruction by pressure, most probably by a benign tumor. The jugular bulb is most probably involved by the tumor. We can definitely say that the VIIIth cranial nerve is involved, as are the nerves above or posterior to the VIIIth. This is a nasopharyngeal lesion which has invaded the posterior fossa by destruction of the occipital bone and the petrous pyramid. There are no signs of increased intra-cranial pressure or of pathological calcifications," Dr. Léger (see Fig. 4).

Surgery was performed under general penthothal anesthesia on May 11, 1954. Through a large horizontal incision the mass was exposed after mobilization of the flaps and the sterno-mastoid muscle, which was reflected backward. Overlying the tumor a small lymph node was removed and found to be normal on frozen section. After exposing the mass it

was obvious that we were dealing, not with a mixed tumor but probably with a glomus tumor, because of its locality and its very marked vascularization. The lower limit of the mass was at the level of the carotid bifurcation, and superiorly it extended to the base of the skull, where a round smooth erosion measuring about 1½ cm. in diameter could be felt with the examining finger. The tumor appeared to have originated

Fig. 4. X-ray of the base of the skull shows a smooth erosion of the right petrous pyramid at the level of the jugular foramen. The internal auditory meatus has been completely destroyed.

posteriorly to the internal carotid artery, around which it had extended and surrounded it laterally and partially covered its anterior walls. It was very closely connected to the artery by numerous fibro-vascular bands, which took on pedicle-like proportions in its postero-inferior, inferior and antero-superior aspects.

By a rather laborious blunt and sharp dissection, always attended by considerable oozing, the mass was finally liberated, except for its antero-superior attachments. Prolonged efforts to free the mass at the base of the skull by finger dissection being unsuccessful, a tonsil snare was carefully

inserted as high as possible and the tumor was snared off. Examination of the specimen revealed an intact capsule. During the operation, which the patient withstood without ill effects, 1000 cc. of glucose and 1000 cc. of blood were given. The post-operative course was essentially uneventful and the patient was discharged from the hospital one week after surgery. Pathology reported a softish, flattened 2.5x5x4 cm. well encapsulated mass, which histological section revealed to be a carotid body tumor (see Fig. 5).

Fig. 5. The surgical specimen measured 2.5x5x4 cm., and was surrounded by an intact capsule.

Three months after operation the patient reported some improvement in the movements of his arm, but all other paralyses remained the same. There was nothing to note on periodic check-ups until October, 1957, at which time he reported that a right facial paralysis had gradually set in during a period of one month.

The patient was admitted to the hospital for further investigation. Externally, there was no evidence of recurrent tumor mass. X-rays of the base of the skull showed complete destruction of the right petrous pyramid, extending to its

tip and probably also involving the floor of the middle fossa; there was no evidence of pharyngeal or naso-pharyngeal mass. Dr. Rinfret, the neuro-surgeon, who had seen this patient prior to operation three years before, was called in on consultation. Arteriography of the right carotid artery was unsuccessful, but the left carotid artery was injected with

Fig. 6. X-ray of the base of the skull, showing complete destruction of the right petrous pyramid.

Hypaque and showed nothing abnormal. There was reflux of the dye into the right carotid and it was concluded that this one was probably occluded at the level of the petrous bone (see Figs. 6, 7).

On October 18th, a ventriculography was unrevealing, and it was thought that the destructive process involving the petrous bone did not extend intra-cranially. A right sub-

occipital craniotomy was then performed by Dr. Rinfret. After easy retraction of the right cerebellum a very hemorrhagic mass was exposed and as much of it as possible was removed. This tumor proved to be carotid body tumor and showed the same histological pattern as the cervical tumor excised three years before except that in the present specimen

Fig. 7. P.A. view showing destruction of the petrous bone.

there was definite capsular invasion (see Fig. 8). The patient never fully regained consciousness following surgery and died seven days later. Post-mortem examination was most revealing. The immediate cause of death was due to an infarct of the left posterior myocardium. Examination of the base of the skull and the brain showed extensive invasion of the petrous bone and the right cerebellum. The diameter of the petrous bone was enlarged throughout, and

Fig. 8. Microphotograph of the intra-cranial tumor shows paraganglionic cells invading the capsule; X48.

Fig. 9. Base of the skull viewed intra-cranially. The right petrous pyramid has been completely replaced by tumor.

grossly appeared to have been completely replaced by tumor. The petrous bone is being processed for serial sections. It was impossible to free the right cerebellum because of its invasion by tumor measuring 5x2.5 cm. The right cerebellar tonsil was deformed and the left one herniated. The bulbar region was normal. Dissection of the right carotid artery,

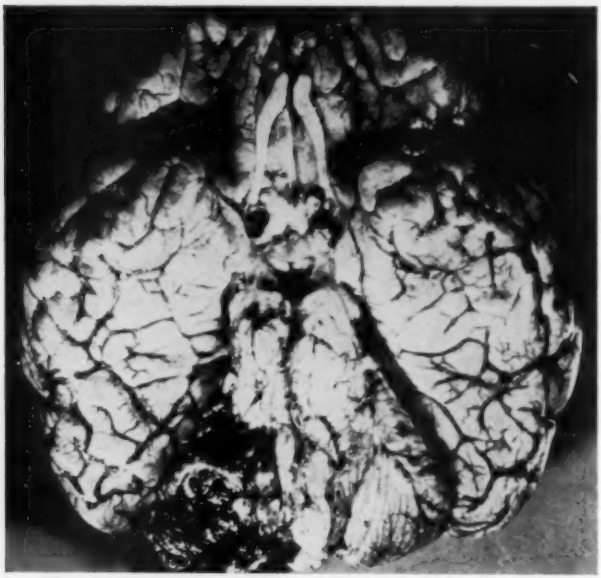

Fig. 10. The brain viewed from below. A large tumor mass has invaded the right cerebellum.

which has been thought to be thrombosed, revealed it to be patent throughout (see Figs. 9, 10).

Many points are of interest in this unusual case. Four years had elapsed from the time the patient first complained of hoarseness to the time the cervical mass was removed.

During this period four cranial nerves became involved as the tumor grew up and caused pressure and erosion at the base of the skull.

The loss of function of the cranial nerves was slow and gradual, as shown by the complete loss of inner ear function without the patient having complained of subjective vestibular symptoms at any time. The cranial nerves were at first probably involved through compression and not infiltration.

Although somewhat tedious, the cervical mass was removed with an intact capsule without injury to the carotid arteries.

Three years later extensive tumor formation was found to have destroyed all the petrous bone and invaded the right cerebellum with a minimum of symptoms.

Both arteriography and ventriculography proved to be most misleading in this case.

The tumor was locally malignant and invasive but did not metastasize. Its growth was slow and destructive and probably extended over a period of more than seven years.

It is difficult to believe that the intra-petrosal and intracranial tumors were an extension of the cervical tumor, as this one had been removed with a complete and intact capsule. Multiple paragangliomas have been reported, and it is not unlikely that in the present case two independent tumors, one arising from the carotid body and one intra-cranial tumor taking origin from intra-petrosal non-chromaffin bodies developed.

Photographs were taken by the Cancer Institute, Notre-Dame Hospital, Dr. L. C. Simard, Director.

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SOUTH CAROLINA SOCIETY OF OPHTHALMOLOGY AND OTOLARYNGOLOGY.

The next joint annual meeting of the North Carolina Eye, Ear, Nose, and Throat Society and the South Carolina Society of Ophthalmology and Otolaryngology will be held in Asheville, N. C., on September 14-17, 1958, at Grove Park Inn. An excellent program is in the making. The following guest ophthalmologists will be present: Dr. James A. C. Wadsworth, New York, N. Y.; Dr. Arthur Gerard DeVoe, New York, N. Y.; and Dr. Frank B. Costenbader, Washington, D. C. The following guest otolaryngologists will be present: Dr. F. W. Davidson, Danville, Pa., and Dr. Tom Rambo, New York, N. Y. A third otolaryngologist will be announced in the near future. A good attendance is anticipated.

For further information write directly to Dr. George Noel, c/o Cabarrus Bank Bldg., Kannapolis, N. C., or Dr. Roderick Macdonald, 330 East Main St., Rock Hill, S. C.

OFFICE CLOSURE OF TYMPANIC PERFORATIONS: A NEW APPROACH.*

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There has been considerable increase in interest concerning closure of central tympanic perforations in the past several years. The advantages of closing a perforation are as follows:

1. Improvement in hearing is usually obtained. The degree of improvement to be anticipated can be estimated by comparing the initial audiometric test level with that obtained by placing a paper patch over the perforation. In some instances the hearing level obtained by closure exceeds that obtained by patch application.

2. The patient can tolerate getting water in the ear—permitting swimming, showers, etc.

3. Recurrent otorrhea is unlikely to occur during respiratory infections.

Closure treatment should be attempted in all dry central perforations in which a complete marginal rim exists. The ear should be dry for several weeks before closure is attempted. There should be no evidence of chronic middle ear or attic disease such as granulations, cholesteatoma debris or squamous epithelium invasion on the promontory. The Eustachian tube should be inflatable.

Older methods of perforation closure consisted of repeated marginal chemical cautery and the application of some type of patch. In 1949 Dunlap and Schuknecht[1] reported their results in treating central perforations by means of repeated cautery and the daily instillation of a few drops of saturated solution of urea. Of 20 perforations so treated, 11 had been closed with an average of 18 treatments each. Six were still

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under treatment and three failed to show any evidence of healing. In 1953 Derlacki[2] reported a modified version of a method of treatment originally described by Linn.[3] He reported approximately 70 to 75 per cent closures. The average number of treatments for those in which closure was accomplished was 14.6. Myringoplasty[4] techniques have been evolved in the past few years, because office closure treatment was rather prolonged and, even if continued a year or two, still left a sizeable number of failures.

Dunlap and Schuknecht[1] describe the histopathology at the perforation margin as follows:

"The process of drum repair stops when the more hardy, rapidly growing stratified squamous epithelium of the external surface of the drum grows over the perforation edge and meets the pseudostratified columnar or cuboidal mucous membrane of the middle ear. Destruction of the perforation margin with acid removes this epithelial barrier and, by tissue destruction incites an inflammatory healing response in the drum margin. Fibroblastic proliferation in the middle layer of the granulating margin is responsible for the laying down of interlacing fibers of collagen. The epithelium grows over the perforation in four days to a week, and must be repeatedly destroyed. When collagen fibers are completely formed they shorten. The shrinking of this circular band of interwoven collagen fibers may facilitate closure by purse string action."

Two clinical facts have been known for many years concerning the healing of tympanic perforations. The first is that many fresh traumatic perforations will frequently heal rather promptly. Second, healing is sometimes accelerated if there is some secondary tympanic infection. It, therefore, seemed logical that closure treatment should incorporate these two basic principles, *i.e.*, maintaining a raw, traumatized perforation margin and, by means other than infection, producing the equivalent of an inflammatory reaction. The objection to using repeated chemical cautery, once the healing process is initiated, is that the proliferative margin is partially destroyed each time cautery is applied. For this reason it

was decided to rely largely upon marginal mechanical trauma to maintain an active border. The inflammatory reaction is created by having the patient instill four drops of a urea boric acid solution in the ear canal twice daily. Urea solution was first used for this purpose by Dunlap and Schuknecht.[1] Linn[2] used Euthymol as an irritant solution.

The addition of the use of loupe magnification in carrying out marginal treatment enabled me for the first time to appreciate what was taking place at the edge of the perforation. Loupe magnification is absolutely essential. Before any treatment is initiated, the margin of the perforation usually presents a beaded or rounded edge. Whether the stratified squamous epithelium extends beyond the inner edge of the margin cannot be determined on external inspection. Visible decrease in the size of the perforation does not occur until there is hyperemia of the tympanic membrane and a pink granular perforation margin. The outer edge of the perforation then loses the "beaded" appearance somewhat.

I first attempted to produce a traumatized perforation margin by serrating the edge with a myringotomy knife point. Occasional activity was produced, but the results were not superior to the use of cautery. After considerable trial and error, a marginal eversion technique was evolved. Special curettes and hooks are used for this purpose. At the initial marginal treatment the curette edge is placed just within the perforation edge and brought out with fairly vigorous pressure against the margin. The basic objective is to evert the marginal epithelium and leave the substantia propria exposed to start granulations. Perforations which heal with rather vascular granulation formation seem to be less inclined to present an atrophic thin drum when healed than do those which have only minimal granulation formation. In the healing process, the squamous epithelium sometimes seems to grow faster than the granulation tissue and again grows inward over the edge, thereby impeding further closure progress. Granulations then accumulate behind the perforation margin on the tympanic side. Everting the epithelium and "rolling" the granulation tissue outward into the plane of the perforation realigns the tissues so that closure again

progresses. In general, the granulations should be everted rather than cut away. There may be occasional exceptions on which comment will be made under the discussion of cases.

MARGINAL EVERSION TECHNIQUE.

1. Anesthesia.

Adequate anesthesia of the perforation margin is essential, as the marginal manipulations are too painful to be tolerated

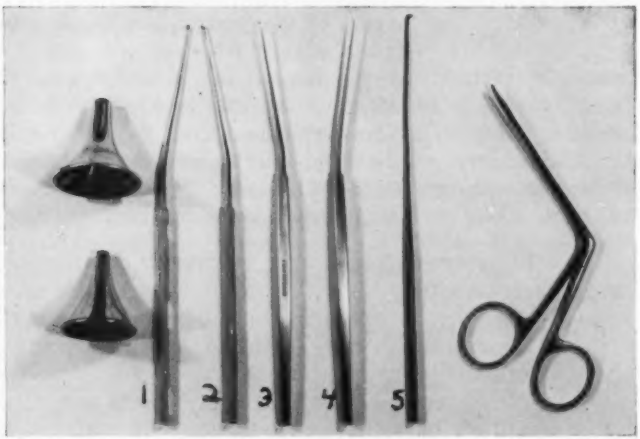

Fig. 1. A wide aperture Boucheron ear speculum is preferred. Intruments Nos. 1 and 2 are special 00 curettes, with special angles to permit an unobstructed view while working. The ring is sharp on both sides (Storz). Nos. 3 and 4 are sharp, right angle hooks (V. Mueller). No. 5 is a Day ear hook. A small standard alligator forceps is used to place the cottonoid patch in the ear canal.

without anesthesia. Block injection anesthesia such as is used for stapes mobilization was not entirely effective and, furthermore, is not practical as a repeated office procedure. After trying various preparations I found Bonain's solution the most effective, but was somewhat hesitant to use this preparation because of its slightly caustic nature; apparently it is well tolerated. Perhaps a minimal degree of chemical irritation accelerates the desired inflammatory action. Just enough is applied with a small cotton-tipped applicator to

cover the perforation margin and the adjacent outer surface of the tympanic membrane. In one instance some of the anesthetic reached the middle ear, and a short period of vertigo followed. Complete anesthesia is difficult to obtain near the malleus and near the tympanic ring.

2. Treatment of Perforation Margin.

On the initial marginal treatment, I have used chemical cautery on only a few. In the majority, the marginal eversion technique was used. In one instance (Ear No. 2) a combination of cautery and marginal curettage was used. The combination may be better as an initial treatment than either method by itself. Further clinical experience will be necessary on this point.

On all follow-up treatments the marginal eversion method was used exclusively as soon as the margin became "active." If the margin has a fine granular appearance and the somewhat gray epithelial layer is not growing inward, only gentle eversion and "freshening" of the granular tissue is carried out with the curette. No attempt is made to cut out granulation tissue. If the epithelium is growing inward over the edge, the margin again has a more firm, "rolled in" appearance, and the epithelial layer is then everted with the curette and hook. Granulation tissue on the inner surface is everted. Some bleeding ensues, but this is not removed, as a blood clot may serve as somewhat of a matrix for connective tissue growth across the perforation.

When the perforation extends to the edge of the malleus, the edge of the pars tensa seems to wrap partially around the malleus handle and is separated from the malleus by means of the curette and hook. This frees the edge of the tympanic membrane and facilitates the start of granulation tissue formation. In ear No. 8 a large granular mass formed around the end of the malleus after a few treatments, and the progress of healing was very rapid thereafter.

3. Placement of Cottonoid® Patch.

In the Linn treatment, a small piece of cotton was fashioned into a pad and placed against the tympanic membrane. It

seemed that the rounded contour of the pad would tend to invert the epithelial edge and impede progress of the granulation activity. Consequently I have used a flat compressed cotton preparation known as Cottonoid.® The commercial preparation is rather thick and is split in half. A piece is cut out to approximate the size of the tympanic membrane. It is moistened with the urea-boric acid drops in order to make the patch more flexible, and is then placed against the drum with an alligator forceps. With a small blunt Day ear hook it is pressed against the entire membrane, care being taken not to push it through the perforation.

4. Irritant Drops.

This solution consists of 2 per cent boric acid and 5 per cent urea in physiological saline. The patient is instructed to instill 4 or 5 drops twice daily. Definite hyperemia is induced. Occasionally the patient will complain of a slight feeling of irritation at first. The urea is the irritant factor, and the boric acid was added to lessen any tendency toward annoying infection.

Mucoid discharge from the middle ear developed in a few instances. This was removed from the middle ear by inflation and aspiration. Treatment of the perforation was continued in these cases, and the added middle ear reaction may have accelerated the rapidity of closure. Should a definite suppurative process develop, then closure treatment should perhaps be temporarily interrupted and the otitis appropriately treated.

5. Interval of Treatment.

Marginal treatment has, in general, been carried out at two-week intervals. Whether this is the optimal interval remains to be determined. The number of cases treated has been too small to vary the technique appreciably. It is possible that inactive margins should be treated more often. The tissues must, of course, be given time to react.

DISCUSSION OF CASES.

The treatment of 15 consecutive central perforations is

summarized in Table I. Each circle represents the tympanic membrane, showing the size and location of the perforation at each treatment. Several significant observations have been noted.

In studying the change in shape of the larger perforations it is evident that the initial closure progress is generally in

NAME AGE	EAR	No.	HEARING BEFORE CLOSURE	HEARING AFTER CLOSURE
T.W. 13	R.	1	8	2
	L.	2	25	5
J.A. 27		3	45	30
H.S. 33		4	38	15
N.K. 38		5	30	8
C.R. 47		6	33	8
T.J. 56		7	33	15
M.S. 43		8	20	3
L.R. 29		9	10	3
J.W. 8	R.	10	22	3
	L.	11	30	
J.W. 24	R	12	7	
	L.	13	7	
E.H. 29		14	17	
O.H. 54		15	62	

TABLE I.

the central portion of the tympanic membrane. A bean shaped perforation assumes an oval appearance. The portion of the perforation margin closest to the malleus shows earlier hyperemia and marginal granulation than does the area adjacent to the tympanic ring. This is undoubtedly due to the fact that the malleolar vessels become hyperemic more readily than do those in the lower portion of the membrane. Healing does not progress until hyperemia is evident.

Ears Nos. 11, 14 and 15 showed early healing activity in the perforation margin nearest to the malleus; however, the inferior portion nearest the tympanic rim continued to show a firm, beaded non-granular margin. At the sixth treatment

in each, the inferior margin of the perforation was cauterized with trichloracetic acid and vigorously curetted. Ears Nos. 14 and 15 are beginning to show granulation activity in the entire marginal circumference following the cautery and curette treatment. Ear No. 11 is still rather inactive, due to the fact that the patient frequently omits the instillation of the drops. On a few occasions the patch appeared quite dry when the patient came in for treatment.

The tympanic membrane in ears Nos. 12 and 13 (patient J. W.) showed a thick pale appearance rather than one of hyperemia. Even though the perforations were small, healing progress has been virtually nil. The epithelium has persistently inverted during the interval between treatments. Though abundant granulation tissue has formed, it has been pale in appearance rather than the usual pink. The tissues present almost an allergy-like reaction rather than an inflammatory one. At the tenth treatment all granulation tissue was removed with a curette and the margin cauterized. At the time of this writing, it is too soon to state whether healing progress will ensue. It may be necessary to change the nature of the drops in this case. Ultimate healing should be possible since the perforations are small.

In summarizing the results obtained by this method of treatment, it is noted that 10 of 15 perforations so treated have to date closed with an average of 4.2 treatments each. Three of the remaining five cases have shown some initial progress (ears 11, 14 and 15) and then reached a standstill. I believe that the reason for this lack of continued progress is due to insufficient mechanical trauma to initiate healing in the inferior margin of the perforation. In the future management of such cases it is planned to use marginal cautery and curettage to the inactive portion of the perforation margin on the second or third treatment if granulation formation is not apparent at this time. An additional reason for slow progress in ear 11 is that this patient did not use the drops regularly. The need for regular use of the drops must be explained and stressed at the beginning of treatment. The exact reason for progress failure in ears Nos. 13 and 14 remains to be determined. Since these perforations are rela-

tively small, it is possible that some variation from the above routine may meet with eventual success.

A few patients with central perforations were not able to come in for regular treatment and were treated with only marginal eversion and the application of a dry paper patch without the use of drops. One healed promptly, and the other is showing satisfactory progress. My impression is that healing in these were more rapid than was noted in the past when cautery and a dry patch were used. These cases are not included in the tabulation.

SUMMARY.

1. A new approach to office closure of central tympanic perforations is presented. It embodies the following changes from previously reported methods:

a. The use of loupe magnification to reveal details of marginal changes during the progress of healing and to permit more precise mechanical technique of marginal traumatizing measures.

b. The use of marginal trauma, rather than cautery, after healing progress has been initiated.

c. Everting the marginal granulation tissue and epithelium.

d. Using a flat "Cottonoid®" patch in order to lessen the liklihood of inverting the marginal epithelium.

e. The use of Bonain's anesthesia before treatment.

2. Ten central perforations have been healed with an average of 4.2 treatments each. The other five cases are still under treatment, and three of these are showing progress.

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PRIMARY CLOSURE OF THE RADICAL MASTOIDECTOMY WOUND; A TECHNIQUE TO ELIMINATE POSTOPERATIVE CARE.*

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It has been said that the history of medicine furnishes no chapter which excels in interest the one devoted to perfecting surgical procedures for the alleviation of suppurations within the temporal bone. To the pioneers in the surgery of chronic otorrhea we owe the fact that the radical mastoidectomy, as practiced today, has reduced the mortality in chronic temporal bone surgery to a minimum; however, the problem of getting a dry, healed ear following this surgery has not been solved by the progress in surgical techniques for the removal of pathological processes. That the problem continues to exist needs no elaboration, for it is common to all who do mastoid surgery.

In a previous paper, entitled "Factors Affecting Healing Following Temporal Bone Surgery,"[1] I pointed out that the temporal bone surgeon, unlike the general surgeon dealing with soft tissue wounds, does not have the crucial advantage of being able to close his surgical wounds primarily. Our two greatest handicaps are: *1.* That our wounds must heal by secondary repair; *2.* that the healing processes are supported by a poor blood supply derived from a bony cavity. These difficulties result not only in a poor quality of healing, but also in a slow process of healing which often allows complicating factors to develop that further delay or completely arrest the progress of repair. I also pointed out that in this course of events the advantage of good temporal bone surgery is often lost because the ear continues to drain.

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Although I had long considered the problem, when the article referred to above was written I did not see the possibility of developing a surgical technique for primary closure of a radical mastoid wound which would eliminate the need for postoperative care. The problem consists of closing an endaural opening with tissue that overlies an air cavity. Free

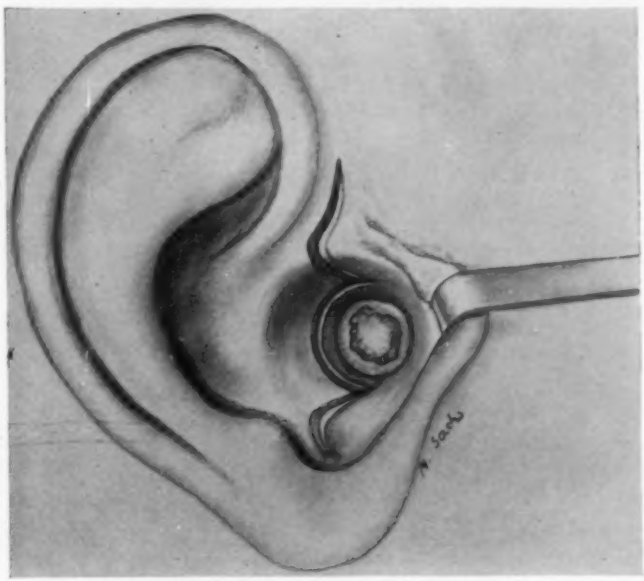

Fig. 1. The endaural incisions consist of superior and inferior incisions from the drum outward, joined posteriorly by a circumferential incision just superficial to the drum.

skin grafts placed in the breech would slough due to the lack of blood supply, and the extent of skin available from the external auditory canal, in any combination of pedicle flaps, is inadequate to close an enlarged endaural opening.

During the past year-and-a-half my experience with the temporal muscle plastic, in the performance of musculoplasty, has suggested a method for primary closure of the radical

mastoidectomy wound. As used in musculoplasty,[2,3] the temporal muscle plastic has solved the vital problem inherent in all reconstructive surgery of the middle ear, that of bridging an air-containing middle ear space. It has accomplished this by reason of the fact that a pedicle flap not only makes available any necessary amount of tissue to cover a large air space,

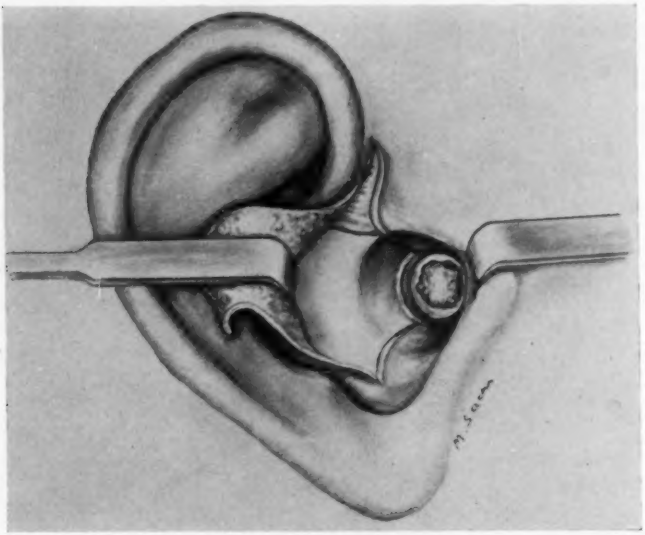

Fig. 2. The skin of the posterior half of the external auditory canal is reflected as a pedicle flap. An elliptical piece of conchal cartilage is removed and the mastoid cortex exposed.

but also carries with it a self-sustaining blood supply; it does not need to obtain blood supply from adjacent structures. The same principle can be used to close an endaural opening.

The technique is described in the legends accompanying Figs. 1 through 9, which follow.

COMMENT.

Now what is actually being done with this technique? It is simply a way to cooperate with nature's efforts to close off

a wound in the temporal bone in the most direct and efficient way possible. It is a natural physiological condition to have a permanently closed air space within the temporal bone open to the pharynx through the Eustachian tube. It is present in the normal ear, consisting of the middle ear cavity and the antrum (see Fig. 10-A).

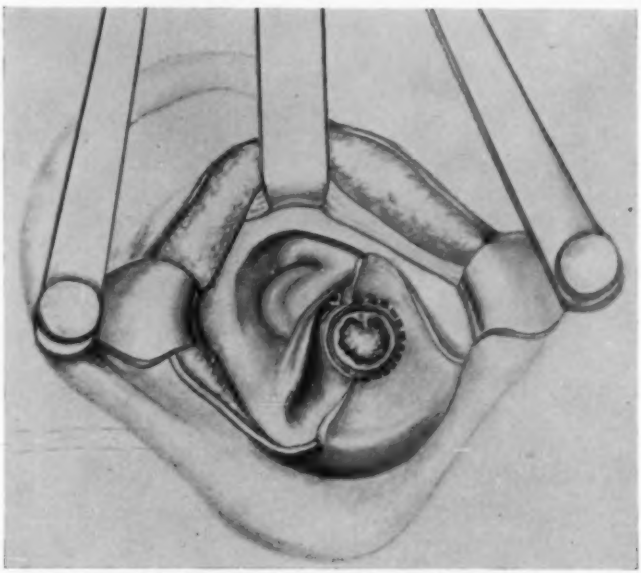

Fig. 3. The mastoid is excavated to remove all diseased tissue. The bony posterior canal wall is taken down; a circumferential incision is then made anteriorly to separate the canal skin from the drum.

After a simple mastoidectomy, with removal of the bone in the mastoid region, a larger space results, consisting of the middle ear, the antrum, and the mastoid cavity (see Fig. 10-B). For many years we have been closing off this enlarged air cavity by primary wound closure with after care that is practically negligible. This has made the simple mastoidectomy the most gratifying operation available today in temporal bone surgery, for both the patient and the surgeon.

With primary closure of the radical mastoidectomy wound we are following the same principle. We simply enlarge the cavity to include not only the mastoid area but also the space previously occupied by the external auditory canal (see Fig. 10-C). It does not matter whether the space is large or small so long as it is connected to the pharynx through the Eusta-

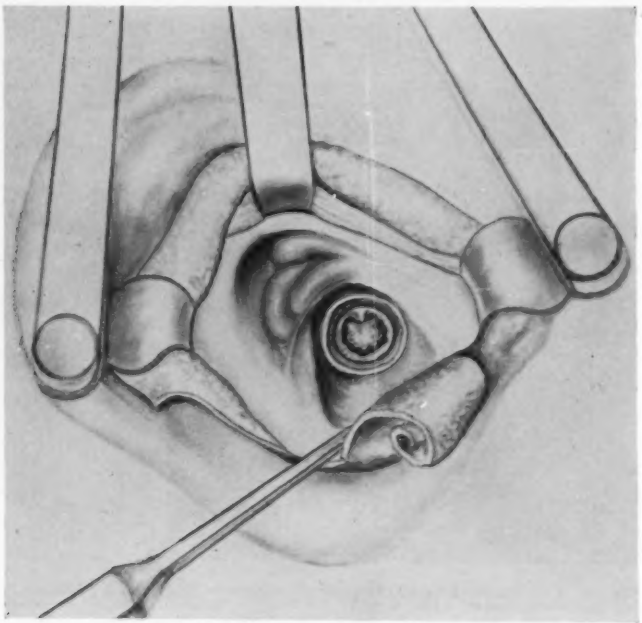

Fig. 4. The anterior canal skin is then reflected as a pedicle flap.

chian tube. In each of these instances of enlarging the normal air cavity in the temporal bone, the space is maintained by air pressures through the Eustachian tube, while mucosa extends itself into the enlarged cavity. Only one thing is essential, a thorough radical mastoidectomy must be performed. In addition to the removal of all foci of infection, particular attention must be directed to the elimination of any type of

squamous epithelium. It is perfectly clear that remnants of cholesteatomatous material cannot be left in a cavity that is to be permanently closed.

This method furnishes a durable healing which will not break down, as it often does in secondary repair when poorly nourished epithelium has grown over bone. With primary

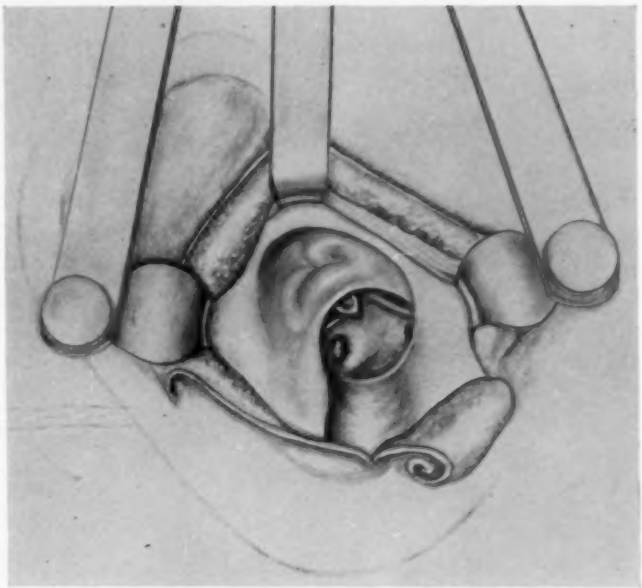

Fig. 5. With the skin of the external auditory canal reflected out of the way as two pedicle flaps, the radical mastoidectomy can then be completed, with meticulous removal of all epithelial and diseased processes.

closure of the radical mastoidectomy wound, patients may take showers and swim. Another important advantage is that there is no postoperative cavity which requires periodic care.

Today, restoration of hearing in deafness, caused by long-standing chronic otitis, is being accomplished. In such cases

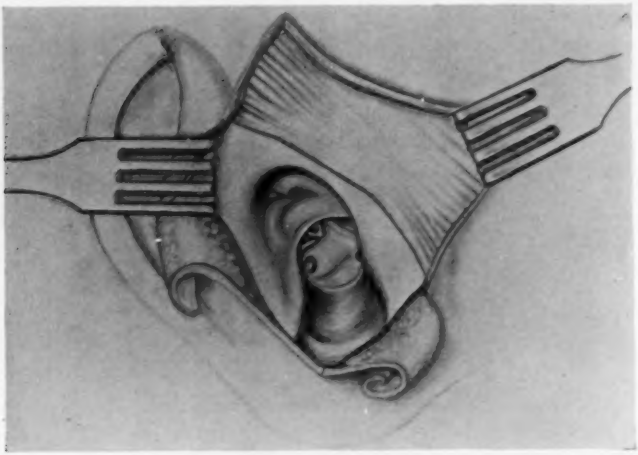

Fig. 6. The temporal muscle is then exposed by extending the superior endaural incision upward and by separating the loosely attached scalp from the temporal fascia.

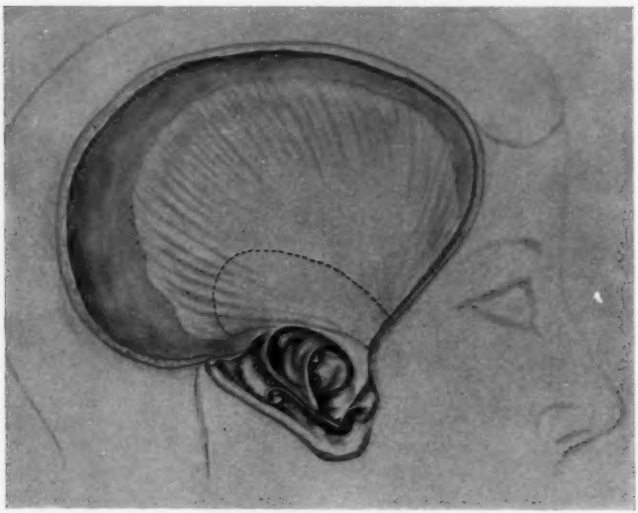

Fig. 7. A temporal muscle pedicle flap is cut as indicated. The flap receives an abundant blood supply, mainly from the posterior deep temporal artery which courses upward and backward in the area of the muscle included in the flap.

an open cavity is essential for the transmission of sound vibrations to the inner ear. Primary closure is not suggested, of course, in these ears, where an open cavity serves a useful purpose; but it is a logical procedure for those patients with poor bone conduction, where an open cavity does not serve any useful purpose. Aside from those in whom bone con-

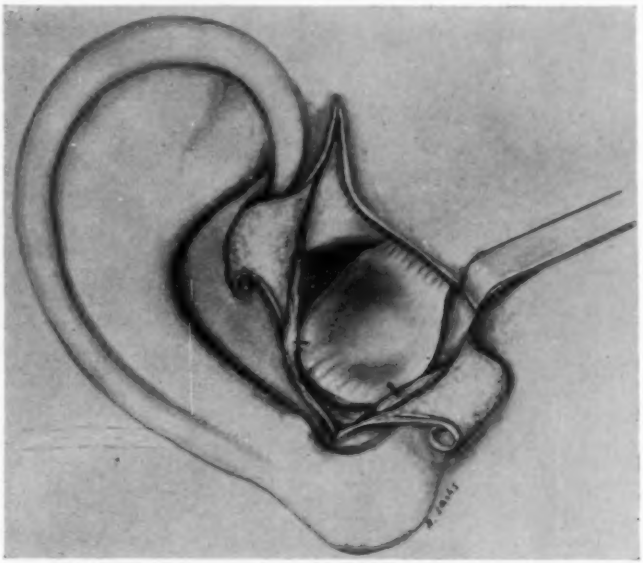

Fig. 8. The temporal muscle pedicle flap is then rotated over the area of the endaural opening. The free end is sutured anteriorly and posteriorly to the soft tissue underlying the skin.

duction is a consideration, there is also a large group of patients with chronic ears who either do not wish to have an extensive plastic reconstructive operation, or for whom such an operation would not be indicated in the best judgment of the surgeon. Among these are the very young, the very old, and those with serious systemic disease. Patients with one normal ear who have no real hearing handicap but who would like to get rid of the basic disease and symptoms, and

get a dry ear in the simplest and easiest way possible, might well be considered logical choices for the procedure.

Even where an open cavity is maintained, the use of the temporal muscle plastic in a postoperative radical cavity—to create a closed middle ear space and to line the cavity with soft tissue which contributes an abundant blood supply—has

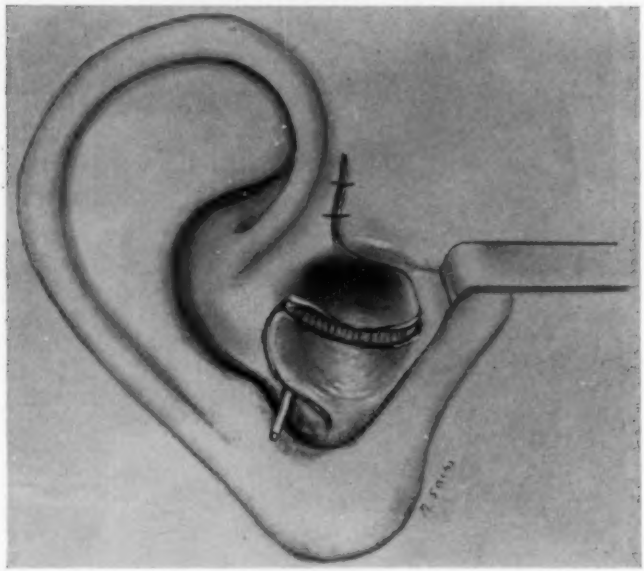

Fig. 9. The two pedicle skin flaps, previously prepared, are then laid in place over the muscle. The superior endaural incision is sutured; a polyethylene tube is left in place for several days as a temporary drain from the cavity.

proved an excellent way of getting prompt healing. The problem of secondary healing is mainly one of blood supply. For instance, we need only to recall how well scalp wounds heal, where there is a rich blood supply. In those radical mastoidectomies, where this technique has so far been used to promote healing, and in one-stage musculoplasties as well as in the first stage of two-stage musculoplasties, the abun-

dant blood supply furnished by the temporal muscle pedicle flap has given a rapidity of healing that has been almost a revelation. It must be pointed out, however, that although this method has provided rapid and uncomplicated healing following the classical radical mastoidectomy, it does not provide all the advantages of primary closure. These are: quick healing by primary intention, the elimination of a cavity which requires periodic postoperative care, and the elimination of restrictions on the part of the patient concerning swimming and taking showers.

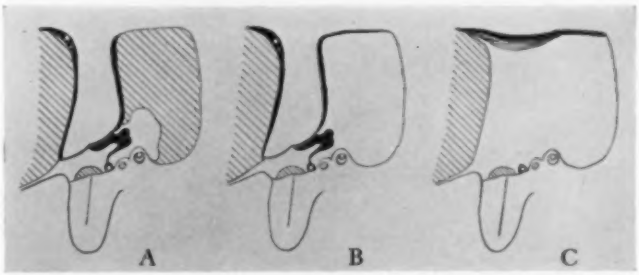

Fig. 10-A. Air space in the normal ear. B—Air space following simple mastoidectomy. C—Air space following radical mastoidectomy with primary closure

Whether techniques are directed toward restoration of hearing, or simply to a cure of the basic disease and attempts to obtain a dry ear, my experience thus far leads me to the conviction that the use of the temporal muscle plastic in surgery of the chronic ear will prove to be the solution to the problem of the persistently draining postoperative cavity.

BRIEF REVIEW OF TYPICAL CASES.

Case 1. A 60-year-old woman with fair bone conduction. In the ear operated she had a large perforation of the drum, destruction of the ossicular chain, suppuration and a large polyp protruding into the external auditory canal. She complained of headaches and some instability of balance. No practical hearing had been present in the ear for ten years, and she had become well adjusted to getting along with the adequate hearing in the other ear. The patient worked in a department store, and she volunteered the information that postoperative visits would be difficult for her to arrange. She was interested only in getting rid of her symptoms, and the chronic otorrhea, with the least possible

trouble. A radical mastoidectomy was performed with primary closure of the wound. Healing was complete, and the patient was discharged after one month.

Case 2. A 46-year-old woman with poor bone conduction in the affected ear. Cholesteatoma was present with destruction of the drum and ossicular chain. Since an open cavity would serve no logical purpose in this case a radical mastoidectomy was performed with primary closure. At the end of five weeks this patient had a dry, healed ear (see Fig. 11).

Fig. 11. Primary healing in a typical case, one month postoperative, is shown.

Case 3. An 8-year-old boy. Cholesteatoma was present in the affected ear after a long history of ear trouble with repeated antibiotic treatments. Removal of the soft, infected cells in the mastoid area, which are typically found in these ears, left a huge cavity after the radical mastoidectomy. In my experience this type of large cavity, in children, has proven to be extremely difficult to heal. With the primary closure technique the healing was complete in four weeks. As is natural for a boy of his age, he is very much interested in swimming. His parents have been told that he may swim without concern for his ear.

Case 4. A 33-year-old business man who came to New York from South America for revision of a radical mastoidectomy which had been performed in Hungary at the age of six. There had been suppuration from the ear since his childhood. The Eustachian tube was open to the cavity, which contained infected polypi and granulation tissue. Bone conduction was poor, and he had had no hearing in the affected ear since early youth. The patient had normal hearing on the opposite side. The cavity

was revised and a technique for obtaining rapid healing which differs from the three preceding cases was utilized. This consisted of bringing a long pedicle flap, cut from the temporal muscle, down into an open cavity to contribute an abundant blood supply to the healing processes and to bridge and seal the middle ear space. Three weeks after operation the patient was forced to leave the city. He returned seven weeks postoperatively with a healed, dry ear; he required no further care.

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AMERICAN BOARD OF OTOLARYNGOLOGY.

The American Board of Otolaryngology will conduct only one examination in 1958, and this will be October 6-9, 1958, in Chicago, Illinois, at the Palmer House.

For further information address Dr. Dean M. Lierle, Secy-Treas., University Hospital, Iowa City, Iowa.

MIDDLE EAR EFFUSION—SYSTEMIC FACTORS.*

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Danville, Pa.

This clinical entity has many synonyms, including secretory otitis media, non-suppurative otitis media, serous otitis media, catarrhal otitis media and hydrotympanum. It is a common condition; our department treats more than 300 cases each year. There is much disagreement in the literature regarding etiology and treatment. In an effort to clarify my own ideas, I have analyzed the case histories of 100 patients who had non-suppurative fluid in the middle ear demonstrated by myringotomy and aspiration.

SYMPTOMS.

The most common symptom is a conductive-type hearing loss, shown by pure tone audiometry and tuning fork tests. It is important to have bone conduction tests, because there is nothing characteristic about the shape of the audiometric curve by air, but a high tone loss is not uncommon. If the disease is unilateral, the Weber test is referred to the ear with poor hearing. Most of the patients had a blocked or full feeling in the affected ear; the typical history states that the symptoms developed following an acute nasopharyngitis.

Most acute middle ear infections respond promptly to administration of antibiotics, but in many cases hearing loss due to fluid in the middle ear persisted until the fluid was removed form one to six months later. I think the increased number of patients with hearing loss owing to middle ear effusion is due to the fact that too many physicians do not utilize myringotomy, as well as antibiotics, for treatment of acute otitis media. The physician treating a child with ear-

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Fig. 1. Pneumatic otoscope with specula of seven sizes which permit its use in a patient of any age. This is essential for the diagnosis of middle ear effusion.

ache has not done his duty unless, after pain and fever subside, he determines whether or not the hearing has returned to normal.

DIAGNOSIS.

Given a patient with a conductive hearing loss, the physician should suspect the presence of fluid in the middle ear. Inspection of the drum reveals moderate retraction. Often the drum has an amber color. Occasionally a fluid level will be seen, and sometimes one will see bubbles of air in the fluid if it is of the serous type. The most important single diagnostic method, which in my experience is almost infallible,

is use of the pneumatic otoscope, shown in Fig. 1. If there is fluid in the middle ear *the drum moves poorly*, and the degree of impairment of mobility is in proportion to the amount of fluid in the middle ear. If the middle ear contains the thick, gummy type of secretion, the drum is practically fixed. In case of doubt, one should do a diagnostic myringotomy and aspiration, using a small spot suction tip against, not through, the myringotomy incision. I use a 15 gauge 3½-inch needle with the point ground off and a 30-degree bend close to the

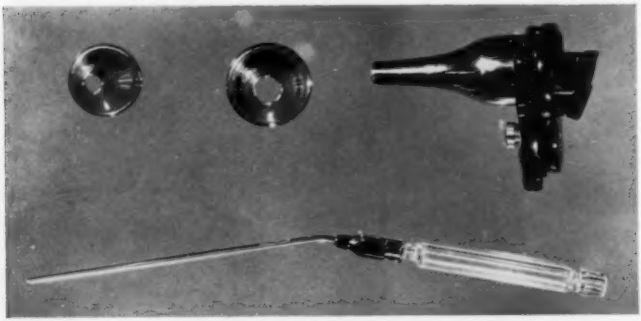

Fig. 2. Aural magnifier, very helpful when doing a myringotomy, and 15 gauge aspirating tip.

hub. This is illustrated in Fig. 2. Aspiration with a 20 or 22-gauge needle inserted through the drum cannot possibly remove the thick, gluey mucus so frequently present in these cases.

In order to use the Siegle type pneumatic otoscope effectively, one must have a speculum that fits the canal snugly. I use specula of seven sizes.

It is not necessary to use a Eustachian catheter for either diagnosis or treatment. If one suspects fluid in the middle ear, diagnostic myringotomy and aspiration should be utilized. If correctly done it is almost painless, and can be done without anesthesia, either general or local, if the doctor has the patient's confidence. One patient had learned how to obtain

relief by puncturing his own drum with the point of a sharpened toothpick.

ETIOLOGY.

I acknowledge with pleasure the fact that my interest in this subject was stimulated by Gordon Hoople's[2] article entitled, "Otitis Media with Effusion; a Challenge to Otolaryngology," published in the May-June, 1950, issue of the Transactions of the American Academy of Ophthalmology and Otolaryngology. Most of the important facts regarding middle ear effusion can be found in Dr. Hoople's article.

I call your attention to one of Dr. Hoople's paragraphs, which reads as follows: "It should not be forgotten that the diagnostic endeavors should be directed not only to the ear, but to the discovery of *general conditions*, which are abnormal."

It seems self-evident that treatment to be effective must be directed at the etiology. It has been said that anything which causes Eustachian tube obstruction, either partial or complete, can cause middle ear effusion, but there are many causes of middle ear effusion and *two or more factors* may be operative in any one patient.

Etiologic factors may be divided into two classes: *1.* Local, and *2.* Systemic. All too frequently we have been concerned chiefly with the local factors, especially hyperplastic lymphoid tissue.

My experience agrees with that of Baron[1] who says that, "A clean, careful surgical removal of nasopharyngeal lymphoid tissue is better than radiation;" however, in only 50 per cent of these 100 cases was careful adenoidectomy a complete answer to the problem. Eleven of these patients, after coming to us for treatment, required a second adenoidectomy, and one required a third.

The patient who has only one or two recurrences of middle ear effusion per year when he has an acute upper respiratory infection, has no serious problem. The patients whose charts

I analyzed had two or more recurrences per year. The apparent etiologic factors are listed in Table I:

TABLE I.—APPARENT ETIOLOGIC FACTORS.

	Cases
1. Hypertrophied adenoid or recurrent lymphoid tissue in the nasopharynx	53
2. Recurrent acute nasopharyngitis (Seven of these had hypogammaglobulinemia)	36
3. Allergy	18
4. Hypothyroidism	11
5. Obesity	9
6. Carcinoma of the nasopharynx	1

LARGE ADENOID AND RECURRENT LYMPHOID TISSUE IN THE NASOPHARYNX.

Our experience agrees with that of Perlman,[3] who stated that there is no correlation between the size of the adenoid mass and the presence of middle ear effusion. His illustrations (Figs. 3-6) show the large amounts of glandular, fatty and lymphoid tissue in close relation with the Eustachian tube, and suggest that an excess amount of any of these tissue elements may produce tubal obstruction. Thoughtful reading of Perlman's article will, I think, discourage anyone from using a Eustachian bougie, or from advising the use of a radium applicator.

Fifty patients who had their adenoids removed stopped having recurrences of middle ear effusion. Ten patients who had recurrent lymphoid tissue in the nasopharynx and recurrent middle ear effusion were treated with X-ray therapy. In nine out of ten cases X-ray therapy failed to prevent recurrent middle ear effusion. It seems obvious that we should consider the *causes* of lymphoid hyperplasia and not confine our efforts to surgical removal or X-ray therapy. Hypertrophied lymphoid tissue, considered as a local factor, may be due to repeated attacks of adenoiditis or nasopharyngitis, but the abnormal susceptibility to infection is often due to a systemic factor, such as adrenocortical insufficiency or hypogammaglobulinemia, so it is very difficult to be sure

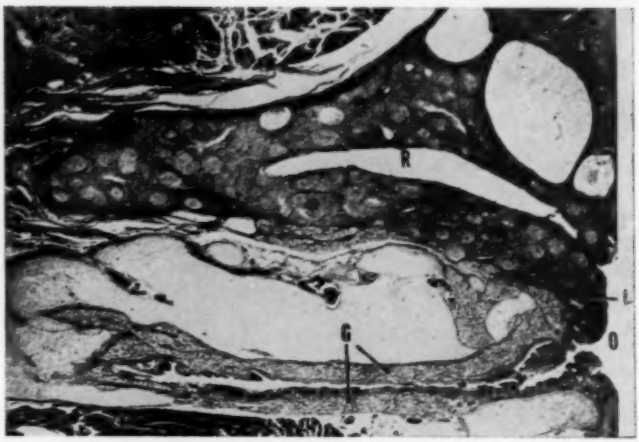

Fig. 3. Section through the mouth of the tube (0), and Rosenmuller's Fossa (R). Note the large number of glands (G) along the tube, and the lymphoid tissue in the Rosenmuller's Fossa, on the torus (L), and at the opening of the tube. (Courtesy of Dr. H. B. Perlman).

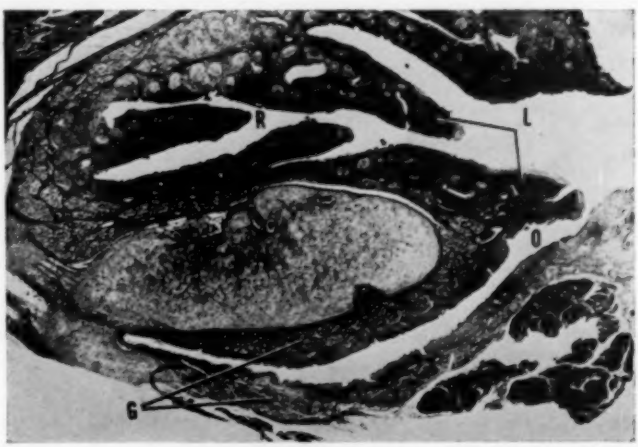

Fig. 4. Shows the tube lined by glands (G), Rosenmuller's Fossa (R), containing large amount of lymphoid tissue (L), continuing over the torus at the opening of the tube (O). (Courtesy of Dr. H. B. Perlman).

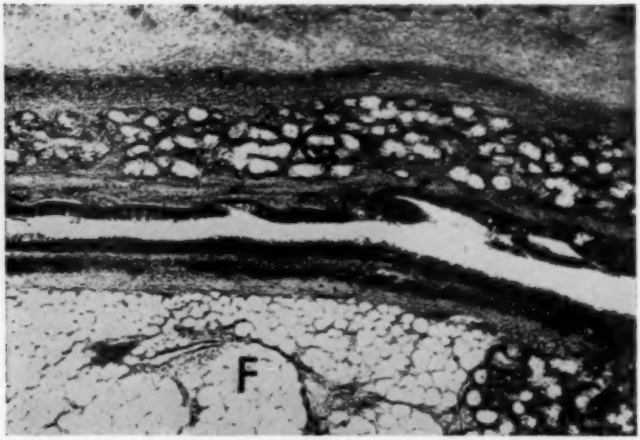

Fig. 5. Photomicrograph through the cartilaginous portion of the tube, showing the large amount of fat and glandular elements along the walls of the tube. These tissue elements are probably important in tubal function, and are particularly resistant to radiation. (Courtesy of Dr. H. B. Perlman).

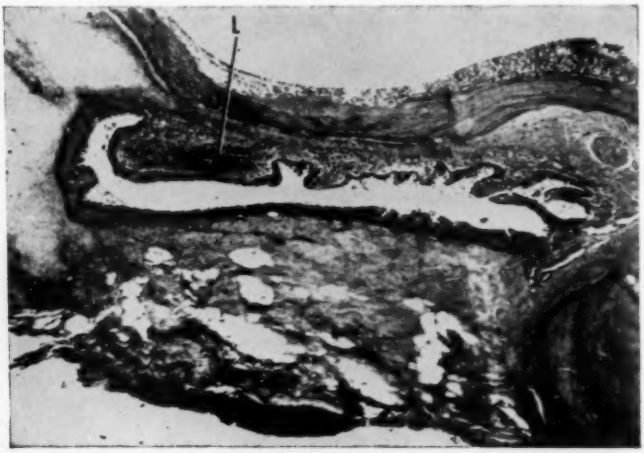

Fig. 6. Section through the Eustachian tube above the torus, showing mucous membrane folds and the subepithelial lymphoid tissue (L), which may influence tubal function. (Courtesy of Dr. H. B. Perlman).

that local factors are strictly local except in those cases in which adenoidectomy stops the recurrences. The age distribution is shown in Table II:

TABLE II.—ONE HUNDRED CASES OF MIDDLE EAR EFFUSION.

	Cases
1—10 years	65
11—20 years	16
21—30 years	6
31—40 years	4
41—50 years	3
51—60 years	2
61—70 years	2
71—80 years	2
Sixty males, forty females	Total 100

This table, showing that 65 per cent of these patients are in the first decade, suggests two etiologic factors:

1. Poor immunity mechanisms, known to be more common in childhood.

2. Hyperplastic lymphoid tissue, known to be more common in childhood.

The influence of hormones on lymphoid tissue is shown by normal involution at the time of puberty. This suggests that the great enlargement of lymphatic structures, frequently seen in early childhood, may be due to insufficiency of steroids, either gonadal or adrenocortical. White[6] points out that there is a marked decrease in the size of lymphoid tissues in response to administration of adrenocortical hormones, and that patients having Addison's Disease have marked enlargement of all lymphoid tissues.

Diet is another factor to be considered. In our experience, a high carbohydrate diet in childhood causes an overgrowth of lymphoid tissue.

Yoffey and Courtice[7] state that the total amount of lymphoid tissue in the body is about 1 per cent of the body weight, so in removing tonsils and adenoids we are removing only a small fraction of the total lymphoid tissue.

RECURRENT ACUTE NASOPHARYNGITIS.

This group of 36 patients is composed of those who had previously had an adenoidectomy, but who continued to have attacks of middle ear effusion each time they suffered an acute upper respiratory infection. This group might well be called the "infection-prone" group of patients, because they had from three to twelve upper respiratory infections per year, often with high fever and sometimes with complications. It seems obvious that there is some systemic deficiency which makes individuals in this group abnormally susceptible to infection. After we began looking for it, we found seven individuals in this group who had hypogammaglobulinemia, according to our standards. This diagnosis will bear some discussion, because there are those academic purists who do not use this term unless the Gamma globulin levels are extremely low. According to our laboratory, the normal range of Gamma globulin is 0.7 to 2.1 gm. per 100 cc., as determined by filter paper electrophoresis. The Gamma globulin determinations for these seven patients, all of whom were children under ten years of age, were 0.66 gm. or 10 per cent, 0.55 gm. or 8 per cent, 0.75 gm. or 11 per cent, 0.8 gm. or 12 per cent, 0.54 gm. or 8 per cent, 0.54 gm. or 7 per cent and 0.61 gm. or 10 per cent. A figure frequently quoted in the literature as normal Gamma globulin is 15 per cent of the total serum protein.

According to our standards all of these youngsters were deficient in Gamma globulin. All of them continued to have frequent recurrences of respiratory infection followed by middle ear effusion despite adenoidectomy, vaccine therapy, allergic management, X-ray therapy and multiple courses of antibiotics, *until* Gamma globulin was administered by intramuscular injection once a month in quantities ranging from 3 to 5 cc., depending upon the age of the patient. Our experience suggests that a Gamma globulin level below 1 gm. per cent predisposes to frequent infections, and that these patients benefit by administration of Gamma globulin.

Four of the seven children who had hypogammaglobulinemia also had allergic symptoms. Turner and Dobson[5] reported

that four of their twelve patients having Gamma globulin deficiency were also allergic, and two of these required specific allergic therapy in addition to Gamma globulin in order to control the main presenting symptoms. This is a clearly written concise article, which helped to focus my attention upon the importance of recognizing Gamma globulin deficiency in patients who are infection-prone.

There are, of course, other factors which cause an individual to be infection-prone, namely overnutrition, undernutrition, poor adrenocortical function, poor thyroid function and deficiencies in the properdin system,[4] about which we are just beginning to learn.

NASAL ALLERGY.

The 18 individuals in this group had either food or inhalant sensitivity or both, demonstrated by history, skin tests and response to treatment. All of the patients in this group had had previous adenoidectomy but continued to have recurrence of middle ear effusion until the allergic factor was recognized and treated by either avoidance of hyposensitization.

HYPOTHYROIDISM.

The group of 11 patients with clear-cut hypothyroidism all responded well to administration of either dessicated thyroid or to triiodothyronine. The diagnosis was suggested by the usual signs and symptoms, and was confirmed by laboratory tests showing low protein bound iodine and high serum cholesterol levels. One patient, a woman aged 67, who had a very large adenoid which Dr. Cole, my associate, removed, had a PBI of 3.03 mcg. and a cholesterol level of 302 mg. per cent. She responded well to adenoidectomy and administration of dessicated thyroid. She had previously been told to get rid of her dog and parakeet because the edematous state of her nasal mucosa suggested an "allergic" etiology.

The X-ray bone age is useful for diagnosing hypothyroidism in children. One youngster, aged nine, had been wearing a hearing aid for three years. Her X-ray bone age was reported as six years. Bilateral myringotomy and aspiration

promptly restored her hearing. Her thyroid deficiency was at first corrected by triiodothyronine and then dessicated thyroid was used as maintenance therapy. Her hearing gain has been maintained.

OBESITY.

Patients in this group of nine continued to have middle ear effusion until they could be induced to reduce their weight by means of low carbohydrate diet. The obese person has excess fatty tissue compressing the lumen of the tube. The overweight factor can be readily overlooked unless the doctor is willing to take the height and weight of the patient and compare it with the height weight table. This simple measure should not be beneath the dignity of any otolaryngologist who considers himself a physician. None of these nine patients was demonstrably hypothyroid, but four of the 11 hypothyroid patients were obese, and carbohydrate restriction was urged in addition to administration of thyroid.

TUMOR OF THE NASOPHARYNX.

There happened to be only one patient in the total number of 100 who had a carcinoma of the nasopharynx. This case reminds us that we should be alert to suspect tumor of the nasopharynx in any adult who has middle ear effusion.

SUMMARY AND CONCLUSIONS.

1. Middle ear effusion is a common condition.

2. A Siegle type pneumatic otoscope is the most important diagnostic instrument.

3. Recurrent nasopharyngitis and hyperplastic lymphoid tissue in the nasopharynx are the apparent local etiologic factors in most cases.

4. Recurrences will be frequent until the systemic deficiencies responsible for the infection-prone status are corrected. One of these frequently overlooked is Gamma globulin deficiency.

5. Recurrences will be frequent until the systemic causes of lymphoid hyperplasia are found and corrected.

6. A careful, visually guided surgical adenoidectomy stops recurrences in only 50 per cent of these cases.

7. X-ray therapy to the nasopharynx stopped recurrences in only one of ten cases treated.

8. Multiple etiologic factors are present in at least 50 per cent of these cases.

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TECHNIQUE OF EXTENDED FRONTO-LATERAL PARTIAL LARYNGECTOMY.*†

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Until recent years, the procedure of laryngofissure (thyrotomy) was the only type of partial laryngectomy widely used; however, it is now quite well established that certain lesions, formerly thought unsuitable for any procedure less than total laryngectomy, because of location or extent, may be treated by other forms of partial laryngectomy without appreciable increase in recurrence rate.

In the type of laryngofissure commonly used, excision of one cord, ventricle and portion of ventricular band is accomplished following sub-perichondrial separation of these structures from the inner surface of one thyroid ala. Portions of the corresponding thyroid ala have at times been removed simultaneously, since this has appeared to favor return of voice (Jackson[4]). The "anterior commissure" technique of Jackson has been useful in cases where resection of the anterior portions of the contra-lateral cord and ventricular band in continuity has seemed advisable; however, in these circumstances, present trends in technique favor removal of an anterior vertical segment of thyroid cartilage, left attached to the soft tissues being removed.

This variation in technique is exemplified by the operation of Clerf,[1] the "bi-lateral thyrotomy" of Kemler,[5] and the "fronto-lateral" operation as described by Leroux-Robert.[6] The latter term seems to be an accurately descriptive and desirable one. All of these techniques are advantageous in avoiding the hazard of inadvertent division through the anterior commissure and/or the lesion, which may occur when a

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mid-line approach through the thyroid cartilage is used, and in providing for greater accuracy in dividing the contralateral cord and ventricular band; furthermore, removal of the anterior segment of thyroid cartilage in continuity provides an additional margin of safety in lesions approaching or beginning to cross the anterior commissure.

The term "extended fronto-lateral" seems appropriate in describing an adaptation of this technique to lesions of somewhat greater extent, such as those showing, *a.* posterior extension to or beyond the tip of the vocal process, but not into the posterior commissure; *b.* beginning impairment of motility without actual fixation of the cord; *c.* limited and superficial extension to ventricle or margin of ventricular band; or *d.* subglottic extension amounting to no more than a few millimeters. Such lesions are removed *en bloc*, along with a large portion of thyroid cartilage with or without the entire arytenoid; on occasion a horizontal or vertical segment of the cricoid may be included.

Post-operative webbing and stenosis are minimized by careful forward suturing of the mucosal edge on the contralateral side, the use of a mold (foam-rubber covered with latex, or acrylic), or immediate skin grafting with insertion of a latex-covered foam-rubber mold. The principles in the latter are those described by Erich[2] in the treatment of chronic laryngeal stenosis and subsequently by Figi[3] in the treatment of carcinoma.

Obviously careful evaluation of the gross extent of the lesion is required before a decision can be made as to the most suitable type of operation. Motility is best studied by indirect laryngoscopy. On direct laryngoscopy, the use of a forward telescope is helpful in determining the antero-posterior extent (magnified image); the for-oblique and right-angle telescopes, along with planigraphy, are useful in detecting sub-glottic extension.

TECHNIQUE.

1. Anesthesia. Initially, procaine 1 per cent infiltration of

subcutaneous tissues in midline from hyoid to supra-sternal notch.

2. Vertical incision in the mid-line from hyoid to supra-sternal notch. Exposure of trachea (by division of thyroid isthmus if necessary), tracheostomy and insertion of endo-tracheal catheter with inflatable cuff well below the cricoid level. General anesthesia is then induced (pentothal and nitrous-ether-oxygen).

3. Vertical division of thyroid ala on contra-lateral (un-involved or least involved) side, 6 to 12 mm. from the mid-line. This is accomplished with the circular saw, attempting to divide only cartilage or cartilage and inner perichondrium.

4. Vertical division through crico-thyroid membrane, contra-lateral cord and ventricular band. This may be along the same line as the division of the cartilage or further posterior, in which case limited dissection beneath the perichondrium underlying the mid-portion of the cord and ventricular band is first required.

5. The lesion is carefully inspected and its gross limits verified.

6. External perichondrium is separated from outer surface of thyroid ala on the involved side, as far as the posterior border and from upper to lower borders.

7. Inferiorly, the line of excision is usually carried along the upper edge of the cricoid arcus and as far posterior as the crico-arytenoid joint capsule, which is incised if the arytenoid is to be removed. The inferior cornu of the thyroid ala is separated from its attachment to the cricoid, on the outer surface of the lamina. During this part of the dissection care must be exercised to avoid entering the thin-walled pyriform sinus. A horizontal segment of the cricoid arcus and lamina may be included if necessary.

8. Superiorly, the line of excision is usually at the upper border of the thyroid ala, although a horizontal segment of the upper portion of the ala is often left *in situ* for support. The soft tissues are divided along a line downward and pos-

teriorly through the upper portion of the ventricular band to the arytenoid.

9. Posteriorly, the excision is completed by extending the superior line of excision downward behind the arytenoid (if the arytenoid is to be removed) or through the base of the vocal process. Strong forward traction (the crico-arytenoid joint capsule and inferior cornu having been previously in-

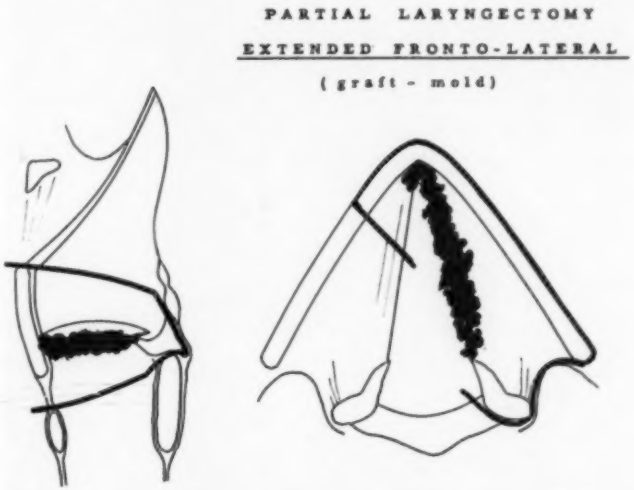

Fig. 1. "Extended frontol-lateral" type of partial laryngectomy. Usual lines of excision are shown in saggital and horizontal planes.

cised) will facilitate inclusion of an adequate margin of normal tissue posteriorly when the arytenoid is to be removed in continuity.

10. The type of repair is naturally determined by the extent of excision required: *a.* If less than one-third of the contralateral cord and ventricular band have been included in the excision, the mucosal edge on this side is anchored forward by horizontal mattress sutures of No. 00000 chromic catgut brought through the external perichondrium of the remaining portion of thyroid ala. A cylindrical foam-rubber mold

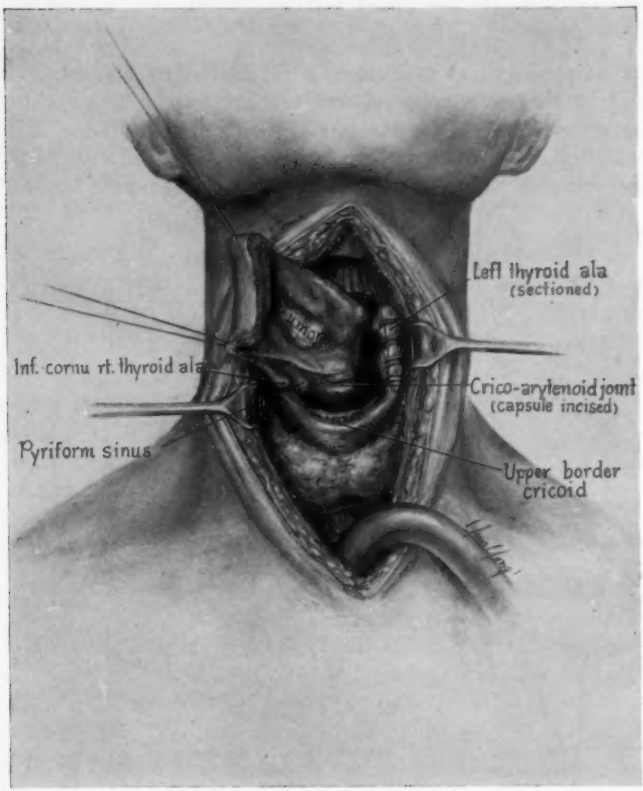

Fig. 2. "Extended fronto-lateral" technique of partial laryngectomy. Left thyroid ala, cord and ventricular band have been divided and the mucosal edge on this side anchored forward by mattress sutures of No. 00000 chromic catgut brought through external perichondrium. On the right, the inferior and superior lines of excision have been completed; the capsule of the crico-arytenoid joint and the attachment of the inferior cornu of the thyroid ala have been divided.

covered with latex (condom rubber) and long enough to extend from the tuberculum of the epiglottis to the first tracheal ring is introduced and anchored in place by two through-and-through sutures of No. 32 alloy steel wire. Laterally and anteriorly, the mold is covered by the external perichondium of the ala which has been removed. *b.* If greater portions of

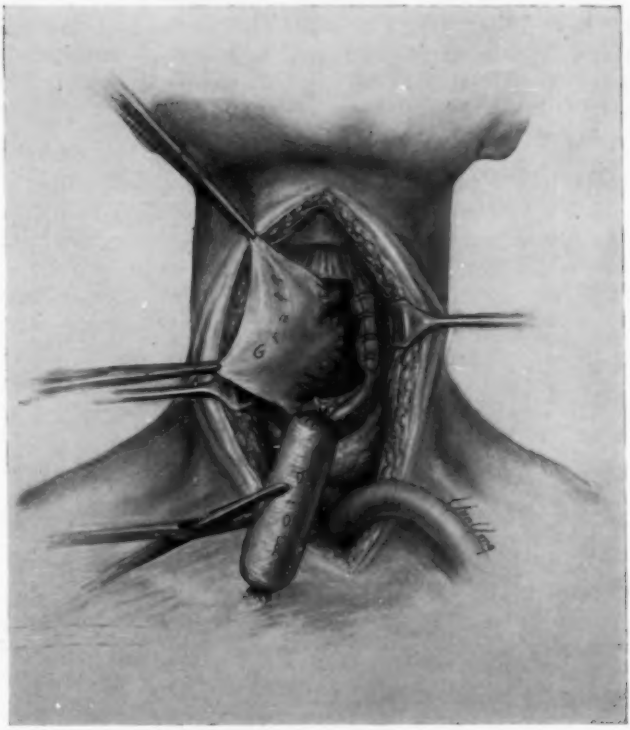

Fig. 3. "Extended fronto-lateral" technique of partial laryngectomy. Placement of split-thickness skin graft is shown. Following introduction of the latex-covered foam-rubber mold, the free anterior portion of the graft is brought across the anterior surface of the mold and sutured to the external perichondrium of the remaining portion of left thyroid ala.

contralateral cord and ventricular band have been removed, or if the arytenoid on the side of greatest involvement has been included, a split-skin graft about 0.015 inches in thickness should be used. This is obtained with the Brown electrodermatome, usually from the area just below the clavicle, and is sutured to the mucosal edge posteriorly, above and below on the side from which the thyroid ala has been removed. Anteriorly, after repair of the mucosal edge on the contralateral side as above, or suture of a second portion

of graft in the same manner, the remaining free edge of graft is brought across the anterior surface of the mold which has been introduced and anchored as above, being sutured to the external perichondrium on the contralateral side.

11. The endotracheal catheter is removed, a No. 6 tracheal cannula introduced, and any accumulated tracheobronchial

Fig. 4. Representative surgical specimen removed by the "extended fronto-lateral" technique. On the left are seen the edge of the divided left thyroid ala, with corresponding portions of left cord and ventricular band. On the right are the posterior border of the lower two-thirds of the right thyroid ala, with its inferior cornu. The entire arytenoid is included, the articular facet being well shown. The lesion involves the anterior commissure and entire length of the right cord, with extension into the ventricle; moderate impairment of motility had been observed. Patient living and well after five years.

secretions aspirated. In closure, drainage is not used, except for that provided around the tracheal cannula, where the edges of the incision are brought together rather loosely.

POST-OPERATIVE CARE.

The usual measures in the care of the tracheostomy patient are employed (humidification and catheter aspiration of

secretions well below the end of the tracheal cannula). If secretions are tenacious, sterile saline 3 to 5 cc. is instilled through the cannula before each aspiration. Nebulization of detergents such as Alevaire® or mucolytic agents and instillation of gomenol 10 per cent are useful in prevention of crusting. The nursing staff should be instructed to report promptly any degree of dyspnea, however mild.

With the mold in place, taking of nourishment does not appear to be an important problem. Liquids are given for the first 24 to 48 hours, following which soft diet is usually well tolerated. Antibiotics (penicillin or penicillin-tetracycline) are given until the likelihood of wound infection has passed, ordinarily about seven to ten days. Pre- and post-operative cultures are useful in determining antibiotic susceptibilities, particularly if the hemolytic staphylococcus aureus, coagulase-positive, should be involved. Demerol or codeine may be used for local discomfort, but are rarely required after the first 24 hours. Ambulation is encouraged on the first post-operative day.

The tracheal cannula is changed daily after the first day and the foam-rubber mold removed in about ten days. Following removal of the mold, some spill-over of liquids into the larynx may be expected for a few days, and aspiration through the tracheal cannula is continued during this period as needed, to prevent the occurrence of aspiration pneumonitis. Once swallowing can be accomplished without appreciable spill-over, the tracheal cannula may be removed, providing the airway seems adequate, after a trial period of corking for one or two days.

SEQUELAE.

As in other types of partial laryngectomy, the occurrence of excessive granulations may require direct laryngoscopic removal, although this is not likely if the skin-graft remains viable. Sequestration of cartilage fragments has not been observed in any of our cases operated by this technique.

Adherence of secretions to the area of skin graft has oc-

casionally been noted for a few weeks or months after operation, but may be largely avoided by bedroom humidification.

Post-operative stenosis of significant degree occurred in two of the series of 16 cases reported here. Tracheostomy followed by insertion of an acrylic mold without re-opening the larynx for periods of ten and 21 days respectively was

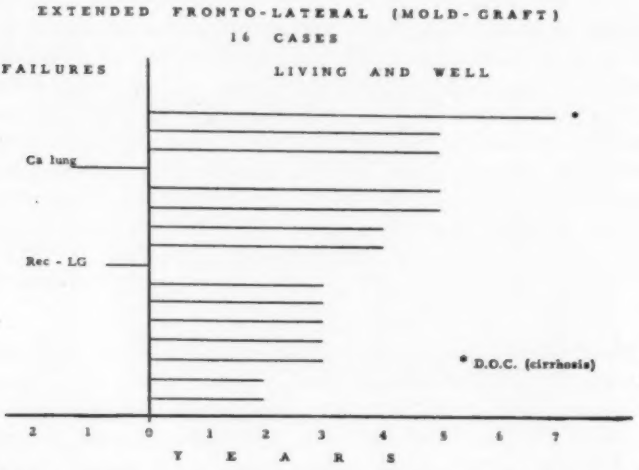

Fig. 5. Results in 16 cases of partial laryngectomy by the "extended fronto-lateral" technique (see text).

required, but in both cases decannulation was later accomplished (in 5 and 12 months respectively).

RESULTS.

Survival data in the 16 cases reported are shown in Table V. Of the six patients who qualify for five-year follow-up, five (83 per cent) are apparent cures and one died 16 months after operation, of carcinoma of the lung, without evidence of local recurrence or cervical node metastasis. Whether the lung carcinoma was primary or metastatic is undetermined.

Of the entire group of 16 cases, only one patient developed

local recurrence. A laryngectomy was done, and he is living and well after four years. There was no operative or post-operative mortality in this series.

COMMENT.

The frequency with which the "extended fronto-lateral" technique may be applied is not great. During the period in which the 16 cases reported were observed, partial laryngectomy by other techniques was performed in 105 cases; *i.e.*, the relative frequency is approximately 1:7; however, no one would question that these patients, most of whom by previous standards would probably have been treated by laryngectomy, have been given an improved outlook for rehabilitation without appreciable increase in risk of recurrence. Although the post-operative voice is, on the average, poorer than following the more limited types of partial laryngectomy, it is usually adequate for ordinary purposes of communication.

As in any type of partial laryngectomy, the initial surgical exposure of the lesion should be through tissues known to be uninvolved, and the actual excision performed without haste, the margin of normal tissue surrounding the lesion being in view at all times. Prompt healing is favored by preserving intact the perichondrium on at least one surface of all remaining portions of cartilage.

Our present technique differs from that of Figi in that, *1.* the split-skin graft is fitted to the mucosal defect and sutured in place, rather than being fixed to the foam-rubber stent which is later to be removed, and *2.* we have not found it necessary to insert routinely a second mold immediately following removal of the first, as described by Figi.

Since all of the lesions treated in this series were essentially of the intrinsic type, or of only slightly greater extent, radical neck dissection was not performed. In none of the cases were cervical nodes palpated initially, and none developed cervical node metastasis after operation.

The "hemi-laryngectomy" of Gluck appears to have been largely abandoned as a form of surgical treatment, at least

in this country. Presumably this is because of the marked post-operative impairment of both the airway and the protective mechanism during swallowing. The term "hemilaryngectomy," which implies removal of an anatomic half of the larynx (including cricoid) as described by Gluck, should probably not be used in referring to the type of operation which we have called, in an attempt to be accurately descriptive, the "extended fronto-lateral."

The usefulness of this procedure appears to be in its intermediate position between the more conservative types of partial laryngectomy and total laryngectomy. By judicious selection of cases, it is likely that one may avoid, on the one hand, the temptation to treat by conservative operation a lesion which is slightly beyond the accepted indications, and on the other, to resort to total laryngectomy when a less radical operation would be sufficient.

SUMMARY.

1. A technique of "extended fronto-lateral" partial laryngectomy is described, and results in a series of 16 cases of carcinoma of the larynx treated by this method are reported.

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CARCINOMA OF THE LARYNX.

Contralateral Metastasis in Lesions Approaching the Midline.*†

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Statistical reports of laryngectomized patients are in close agreement as to the rate of recurrence; approximately 30 per cent of patients thus treated develop metastatic cervical lymph-node disease. Of this group, approximately 35 per cent may be treated successfully by a secondary radical neck dissection. In order to improve our present cure rate, we must consider what happens in these cases which develop cervical lymph-node extension.

It is certain that an improvement may be expected by extending the primary operation to include the lymphatic contents of the neck, but the main difficulty is in selecting the cases which may be expected to benefit by such an extended operation. Kuhn[1] has given us a statistical report on the types of cases where we may expect metastasis to occur. He reported that 57 per cent of all non-cordal and diffuse growths were complicated by metastatic lymph-node disease, and that cordal growths with a moderate extracordal extension ranged up to 34 per cent. Approximately 75 per cent of pyriform sinus and 78 per cent of marginal lesions were accompanied by metastasis.

One cannot help being impressed by the element of unpredictability in the biological behavior of these growths. For example, one patient may have a bulky, primary, extracordal growth without metastasis, and another case with a small cordal lesion may eventually manifest incurable metastatic disease. The exceptional cases should not alter our plan of treatment, which is based on statistical probability;

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but we should recognize at the same time that there is a measure of individual behavior in the activity of the disease, the course it takes, and the response to therapy. We should anticipate and be prepared for the unexpected. It is not possible to make an absolute prediction on the basis of statistics, for no two cases are similar in all of the important particulars.

Successful treatment by means of an extended surgical approach depends not only on anticipating what lesions may produce metastasis but also on which lymph-nodes may be involved.

CLINICAL TYPES OF LYMPH NODE METASTASIS.

The usual case developing clinical evidence of metastatic lymph-node disease does so in the first or second postoperative year. The customary site is in the nodes of the neck on the same side as the primary growth. These cases can be treated with success by a one-stage laryngectomy and ipsilateral neck dissection.

In midline growths, such as those of the epiglottis, anterior commissure, interarytenoid space or postcricoid area, there is obviously a potential for bilateral metastasis, and these cases are much more difficult to manage. Here one is at a loss to predict the direction or directions of neoplastic extension. Until the time when one-stage laryngectomy with bilateral radical neck dissection is more feasible, one must choose either an operation including the laryngeal lesion and the lymphatics of the side of greater involvement, or wide-field laryngectomy alone.

Less frequent and less to be expected is contralateral metastasis from a lesion decidedly on one side but approaching the midline. Reed[2] has reported this to occur, however, in 10 per cent of the cases developing recurrence after laryngectomy. If we include those lesions which produce bilateral metastasis, the figure comes closer to 20 per cent. This contralateral metastasis is an occurrence which laryngectomy and neck dissection on the expected side of metastasis would

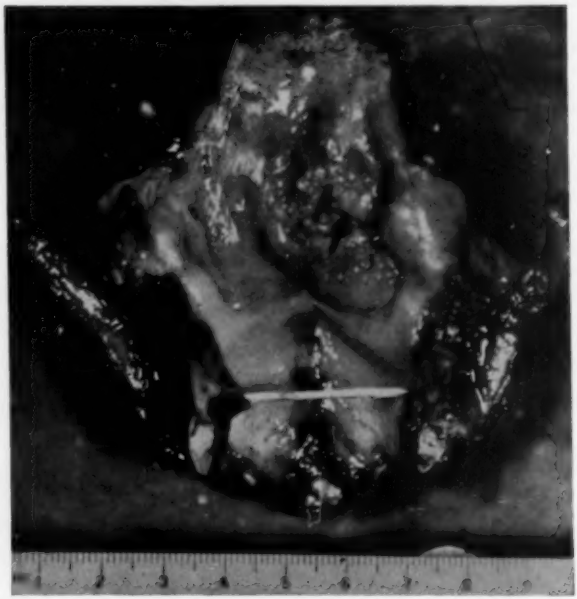

Fig. 1. Photograph of the gross pathological specimen showing a large right epicordal growth involving the ventricular band, base of the epiglottis, and extending to the midline. There is a congenital absence of the greater horn of the thyroid cartilage on the right.

be powerless to cure. I have had two cases which presented this problem.

CASE REPORTS.

Case 1. C.H., a 66-year-old white male, was first seen April 3, 1951, and complained of progressive hoarseness of one-and-a-half year's duration and increasing difficulty in breathing, and pain around the right ear of two months' duration. Examination of the ears was negative. Examination of the larynx showed a large, proliferating, neoplastic lesion of the right ventricular band and base of the epiglottis. The breath space was narrowed so that the true vocal cords could not be visualized. There was no palpable cervical lymphadenopathy.

Tracheotomy, followed by laryngoscopy and biopsy was done. The biopsy report was transitional epidermoid carcinoma, Grade II. Wide-field laryngectomy was performed under local anesthesia on April 30, 1951. The surgical specimen is shown in Fig. 1, and demonstrates a large right ventricular band and epiglottic base lesion, which extends to the midline of the epiglottis. The entire hyoid bone, strap muscles,

larynx and upper tracheal rings were removed. The only other abnormal finding was that there was a congenital absence of the greater horn of the thyroid cartilage on the right.

Convalescence was prompt, and he was discharged from the hospital on the ninth postoperative day. He did not learn esophageal speech, due to a marked nerve deafness, but he progressed satisfactorily otherwise.

On June 20, 1952, two hard, freely movable, metastatic nodules were palpated under the anterior border of the left (contralateral) sternomastoid muscle. The nodules were approximately 2 centimeters and 1

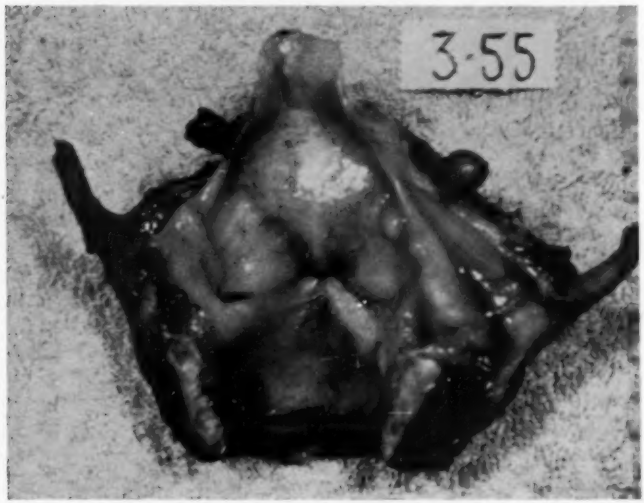

Fig. 2. Photograph of the gross pathological specimen showing a growth of the right periarytenoid region extending into the ventricle. There is a small cyst just below the anterior portion of the right aryepiglottic fold.

centimeter in diameter. Since they were freely movable, and since he was otherwise a good surgical risk, a left radical neck dissection was advised. This advice was declined, and the patient did nothing until July 6, 1953, when he presented himself for radiological treatment. The metastatic neoplastic masses were then large, fixed, and attached to the skin; 8000 *r* were given to the left side of the neck and, as might be expected, the response was not favorable. The metastatic masses continued to get larger, and he died on January 26, 1955. At no time during the three-and-a-half years he was known to have metastatic disease in the left side of the neck did it become clinically manifest on the right side, the side of the epicordal primary growth.

Case 2. H. V. R., an 81-year-old white male, was first seen April 4, 1955, and complained of hoarseness of six weeks' duration. He had been in

good general health most of his life, but had had a "slight stroke" two years before without any residual symptoms. Examination of the larynx showed decreased motility of the right true vocal cord. On a subsequent examination, a diffuse surface irregularity was observed around the right vocal process of the arytenoid cartilage and the posterior portion of the true cord. Laryngoscopy with biopsy was not done until July 15, 1955, at which time there was a diffuse, peri-arytenoid, hemorrhagic, surface lesion extending into the right ventricle. The pathological report was squamous carcinoma, superficial (ungraded). There was no palpable cervical lymphadenopathy.

Wide-field laryngectomy under local anesthesia was done on August 2, 1955. The hyoid bone, strap muscles, larynx and upper tracheal rings were removed. The lesion (see Fig. 2) surrounded the right vocal process, and measured 2x1 centimeters. It appeared to be flat and superficial; however, on microscopic examination there was deep invasion around the right arytenoid cartilage, extending into the deep intrinsic musculature. It approached, but did not reach the midline of the interarytenoid space. The microscopic diagnosis was Grade III epidermoid carcinoma. He was discharged from the hospital on the eighth postoperative day and did well until January, 1957, when a hard, fixed, metastatic neoplastic mass was palpated in the area of the left (contralateral) deep, superior, cervical lymph-node. In view of his age and rather feeble general condition, and in view of the fixation, a radical neck dissection was not advised. He remained comfortable but slowly became worse, and died on July 7, 1957. Although the solitary metastatic mass in the left side of the neck enlarged, at no time was there any clinical evidence of metastatic lymph-node disease on the side of the primary laryngeal lesion.

DISCUSSION.

One could explain contralateral metastasis by the presence of anatomical variations in the lymphatic drainage system; however, Willis[3] has provided us with a pathological explanation, based on the disturbed dynamics of the lymphatic drainage system in the presence of neoplastic disease. He suggests that the volume of primary neoplastic tissue may produce an obstruction in the normal lymphatic flow, forcing retrograde carriage of tumor emboli and crossed deposition to the opposite side. He further states that this crossed metastasis may antedate lymphatic spread to the ipsilateral side. One would expect this to occur in moderate to large volume tumors which approach the midline. This was the situation in both of the cases reported. In the epicordal case, the lesion was large and extended to the midline. In the peri-arytenoid case, the growth appeared to be very superficial; however, on pathological examination it was found to extend deeply around the right arytenoid cartilage, although not reaching the midline posteriorly. In each of these cases, a one-stage laryn-

gectomy, with ipsilateral neck dissection, would have failed to encompass the disease.

SUMMARY AND CONCLUSIONS.

1. A report of two cases of diffuse unilateral carcinoma of the larynx, which produced contralateral metastasis, is presented.

2. These cases confirm the incidence of contralateral metastasis previously reported by Reed.

3. An explanation for this occurrence, based on retrograde embolic deposition, has been provided by Willis.

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AMERICAN ACADEMY OF OPHTHALMOLOGY AND OTOLARYNGOLOGY HOME STUDY COURSES.

The 1958-1959 Home Study Courses in the basic sciences related to ophthalmology and otolaryngology, offered as a part of the educational program of the American Academy of Ophthalmology and Otolaryngology, will begin on September 1 and continue for a period of ten months. Detailed information and application forms may be secured from Dr. William L. Benedict, the executive secretary-treasurer of the Academy, 15 Second Street, S. W., Rochester, Minnesota. Registrations should be completed before August 15.

AN OBJECTIVE APPROACH TO THE COMPLAINT, LUMP IN THE THROAT.*†

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Patients complain frequently of a lump in the throat. This term sums up for them a number of vague, uneasy complaints which are annoying and, sometimes frightening.

The physician in his office, or at the bedside, notes the complaints and clinical findings of his patient, and supplements his knowledge by laboratory studies. With this picture in his mind, he may consult the literature.

Modern systematized medicine usually assumes the diagnosis and recounts the symptoms, signs and laboratory findings accompanying the condition. In many instances, the two pictures are readily superimposed; however, in a complaint such as this, the results are likely to be confusing. Without a definite plan of investigation, the physician frequently succumbs to the easy and popular view, and labels the patient a neurotic.

A few years ago, the author became interested in this complaint. One hundred and seventy-five patients were studied objectively over a five-year period. The results of that survey will follow.

Reference to the medical texts showed this complaint to be associated mainly with functional disturbances. Some authors exercised considerable imagination in determining the causes; others took a more objective approach.

Thomson and Negus,[1] in discussing sensory affections of the pharynx, note that hyperalgesia and paraesthesia may

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lead patients to complain of soreness, irritation, burning, choking, dryness, etc., and a sensation of foreign body in the throat. Most cases, they feel, are found among the hysterical and neurotic, but abnormal sensations are also met in the anemic, gouty, dyspeptic, alcoholic and hypochondriac. The nose, throat, lingual tonsil, ears, teeth, refractive errors, etc., may be a cause of reflex irritation. Lederer[2] says that, in a large proportion of so-called hysterical cases, some local lesion will be found to account for the symptom, if the pharynx is carefully examined.

In the literature, Tremble,[3] among others, directed attention to the lingual tonsil. Mills[4] reported cases in which the nasal sinuses were the cause. Morrison[5] stated that osteoarthritis of the lower cervical spine was a frequent cause of lump in the throat. According to Boreadis and Gershon-Cohen,[6] osteoarthritis in the region of the vertebral foramina, with narrowing and roughening, is very important. Inman[7] believes there are two separate kinds of sensation: one caused by local inflammation over the arthritic process, and the other along the whole length of the peripheral nerve.

Lindsay,[8] in discussing functional diseases of the upper swallowing mechanism, notes that a tonic spasm of the pharyngeal musculature may occur in disorders of the central nervous system, such as tetanus or brain injuries, as well as in acute local infections. A tonic spasm probably occurs also in some psychogenic disorders (cricopharyngeal spasm, globus hystericus).

Pastore, Aldrich, Richards and Lindsay,[9] in a panel discussion on "A Lump in the Throat," covered the organic, psychic and physiological aspects of the subject.

Clerf[10] stated that spasm of the cricopharyngeus frequently may be called a lump in the throat. The latter may be caused, also, by organic lesions in the esophagus or by reflex action from more distant parts, associated with pharyngitis, arthritis, or straight autonomic stimulation. This latter cause was developed by Hilger,[11] who pointed out that disturbances of the autonomic nervous system cause neurovascular changes in a number of areas. These are evident in

the hypopharynx as a pain and a lump. Sjoberg,[12] in experimental work on cats, showed that a high bilateral vagotomy caused paralysis of the vocal cords and pharyngeal paralysis. There was also a strong contraction of the cricopharyngeus and cardiospasm. This, he claimed, was due to a sympatheticotonic contraction at both levels of the esophagus.

A comprehensive study of the group was carried out under a wide variety of headings. These may be arranged as to

TABLE I.—AGE AND SEX OF 175 PATIENTS.

Female	116
Under 40 years of age	65
Over 40 years of age	51
Male	59
Under 40 years of age	19
Over 40 years of age	40

TABLE II.—SITE AND DURATION OF LUMP.

Site of Lump—	
Cricoid Area	107
Vallecular Area	68
Duration of Lump on First Visit—	
Under 1 month	53
Under 2 months	91
Under 1 year	143
After 1 year	32

age and sex, site and duration of the lump, associated complaints, clinical findings, management, treatment and results of treatment. Notes were made on the estimated psychogenic element involved in each case.

It will be noted that two out of three cases in this series were female. Contrary to general opinion, they were more likely to be under the menopausal age. Males with this complaint were more frequently over forty years of age.

The lump was localized most commonly at the cricoid level in the midline. In the remainder of the cases, it was situated higher, usually about the vallecular level, to one side or other of the throat. Most of the patients were sufficiently impressed with their complaint to seek early advice.

Lump in the throat is the expression the patient finds best describes the vague, disturbing sensation which warns him all is not well in this region of his body. The associated complaints may be equally difficult to describe.

In order of frequency, the following symptoms were noted: A feeling of dysphagia, soreness, fullness, and foreign body. Cough, hoarseness and bleeding were present in lesser numbers.

Dysphagia, to the point of actual interference with the swallowing of fluids or solids, was not common, although

TABLE III.—ASSOCIATED SYMPTOMS.

Dysphagia	60
Soreness: Pain	57
Fullness: Pressure	43
Feeling of Foreign Body	28

Less frequently—Cough; Hoarseness; Bleeding.

TABLE IV.—CLINICAL FINDINGS.

Pharyngitis	66
Post Nasal Discharge	37
Cervical Arthritis	23
Esophagitis	21
Malignancy	15
N. Y. D.	30

Foreign body; cysts; elongated styloid process; hyperthyroidism; anemia; alcoholism; tobacco.

occasionally seen with loss of weight. It was usually described as difficulty with a dry swallow—their own secretion.

Soreness and pain were of a vague, nagging nature, usually about the location of the lump. They occurred relatively more frequently in the ventricular area. When present at the cricoid level, they occasionally radiated substernally.

Fullness was usually associated with those who felt a foreign body or secretion might be present in the vallecular area. Pressure was particularly noted in the vallecular area, or in patients with a large thyroid lobe.

In order of frequency, pharyngiitis, post nasal discharge, arthritis of the lower cervical spine, esophagitis and carcinoma of the hypopharynx and upper esophagus, were the most common findings. Found less often were foreign body; cysts, particularly in the vallecula; elongated styloid process; lack of movement of a vocal cord. It was noted in patients with hypothyroidism and sideropenic anemia. Patients with hyperirritable throats, as well as those who used alcohol and tobacco to excess, were noted in the series.

Pharyngitis was the most common finding, noted usually in the lingual tonsil and the region of the lateral pharyngeal bands. Recent or active inflammation, rather than hypertrophy, was the important finding. Frequently, there was an area of hyperemia in the region of the lateral pharyngeal band just medial to the posterior pillar of the tonsil.

In most instances, alleviation of the offending cause by antibiotics or other therapy stopped or relieved the complaint. Patients often stated that previous sore throats had caused the same complaint. Occasionally, post nasal discharge accompanied the pharyngitis, but the two findings were not regularly associated.

Patients with post nasal discharge fell into two groups: those with profuse, and those with dry, crusty secretions, such as are frequently seen with a low-grade posterior sinus infection. Suppurative antral and ethmoidal infections were a factor in a few cases, and treatment of the sinus infection removed the complaint. Patients in the group were temporarily relieved of the lump by swallowing.

A number of patients with obvious allergies or irritations to paints, chemicals and detergents were seen who, at times, had this complaint.

Arthritis of the cervical spine was observed radiologically in a number of cases. The lower three cervical vertebrae were more commonly involved. On the whole, other significant findings were not present, although the patients occasionally complained of soreness in the back and sides of the throat and in other areas, such as the shoulders and arms. Patients with severe arthritis did not complain of a lump in the throat.

A number of patients felt that a foreign body might be present, accounting for their symptoms. Where the history warranted, radiological studies and direct examination were carried out. Sometimes, in indirect examination of the hypopharynx or direct examination of the esophagus, evidence of injury by a passing foreign body could be seen. Only in a relatively small number was a foreign body found. It would seem that, for a foreign body to be interpreted as a lump, it must be small and relatively non-irritating. Injury to the retropharyngeal soft tissues with thickening may, in its early stages, feel like a lump.

TABLE V.—ESOPHAGEAL EXAMINATION. 42 CASES.

Negative		8
Upper Esophageal Cancer		7
Foreign Body Trauma		5
Diverticulum		1
Esophagitis		21
	Cricoid and Cardiac Levels	10
	Cricoid Level	9
	Throughout	2

Two instances will be mentioned briefly to show that these latter factors cannot be ignored. A man was sent to my office with a lump in his throat and an accompanying pharyngitis and "flu" ear. The night before, while eating a green salad, he had a stinging sensation in his throat. Beyond the signs described, nothing further was evident by indirect examination in the region of the hypopharynx. A week later, he returned with a greatly swollen arytenoid and pyriform fossa. A small metal staple, probably the one used to close the plastic bag containing the greens, was seen on X-ray.

The second case was sent to me a week after she had complained of a lump and history suggesting a foreign body. There was no longer the complaint of a lump. Instead, there was a massive swelling of her lower retropharyngeal tissues. A huge, superior mediastinal abscess was drained through the neck.

Forty-two patients were examined esophagoscopically. Early upper esophageal cancer, foreign bodies and foreign

body trauma, and diverticula were found in a small number of cases to be the cause of the complaint.

The remaining cases, constituting half of the group, were labelled esophagitis. They did not include inflammatory changes associated wi the first half of the group. Ten of the cases showed a circular band of hyperemia and esophagitis extending an inch or more down the esophagus from the cricoid level with a similar diffuse, and usually less apparent, area present at the cardia. Nine cases showed esophagitis only at the upper or cricoid level, and included three with web formation. Two cases had more or less diffuse esophagitis throughout with the greatest involvement in the upper area.

The group with esophagitis at the cricopharyngeus and cardia probably represent spasm at the two levels. The second group seemed to be associated with a number of factors, such as reflex spasm, allergic and other irritants, sideropenic anemia.

RADIOLOGICAL FINDINGS.

Many of these cases had radiological studies including the chest, cervical spine, barium bolus and occasionally, spot films. This was carried out in every case before esophagoscopy. Studies of the lateral cervical spine showed the soft tissues and largeal cartilages, as well as the cervical spine. A number of interesting pictures of osteoarthritis were obtained. Barium studies in most cases gave evidence of postcricoidal cancer, although there was a tendency sometimes to miss a fair-sized lesion in this area. Definite evidence of cricopharyngeal spasm was not obtained, although, in a few instances, the radiologist suspected a hesitation in the bolus entering the esophagus.

The management of a patient with lump in the throat is important. With such a vague complaint, the patient frequently suspects the worst. The functional and emotional element is a varying factor in all cases. Nearly everyone, when carefully questioned, can relate stresses and strains in their daily life. In this series, clinical findings of significant value were found in the majority of cases. Treatment of the known

causes, with allowance for the overlying functional factor, should be the keynote of therapy.

The history and examination must be thorough. Treatment will be based on the examination findings. Where there is no response to therapy in a month or so, thorough radiological studies should be made. If the results are negative and supported by clinical findings, a further waiting period is justified. Continuation of the symptoms at the cricoid level or lower should be followed by esophageal examination.

As soon as possible, a confident attitude must be struck, and the patient given to understand that the examiner is familiar with his complaint. He is told that it is seldom serious and that the necessary steps to safeguard his health have been taken. It is well to let him know that, while most patients respond readily, others may have trouble for a considerable period of time. The advice of the family physician is available in most cases; the internist is frequently consulted, the neurologist occasionally, and the psychiatrist rarely.

The treatment of pharyngitis, inflamed lingual tonsils, lateral pharyngeal bands, postnasal discharge, foreign bodies and injuries caused by their passage, need not be labored. Treatment for the hypothyroid and anemic is essential. Advice regarding alcohol, tobacco and obesity is important.

Esophagoscopy is more than a diagnostic procedure. The stretching of the cricopharyngeal sphincter, breaking down of adhesions in arthritis cases, and removal of webs is beneficial. The application of dilute silver nitrate seems beneficial in prolonged esophagitis. In addition to medication for presenting conditions, thiamin chloride is frequently used, sodium iodide for dry throats and, in cricopharyngeal spasm, antispasmodics.

The results of therapy were good in the pharyngitis cases. Most cases fell into the cured or improved group, with few failures. Improvement usually meant that the patient was cured of the complaint, but that it tended to recur with additional pharyngitis. The results were much the same for postnasal discharge.

The arthritis group was much harder to estimate, both as to their validity and the result of treatment. It should be noted that the diagnosis is made, primarily, radiologically. A like group of patients without complaint would, doubtless, show characteristic osteoarthritis. Mostly, they were improved, or no better. About half of the esophagitis cases were cured, the remainder falling about equally into the improved and the no better category.

Patients with a legitimate foreign body or a vallecular cyst treated, did well. The vague foreign bodies and the N.Y.D. group were mostly improved, with some cured and others no better.

Where the lump was an early symptom of carcinoma or a stenosing lesion, the progress of the patient was that of the disease. The complaint, however, became lost in the more serious presenting symptoms.

SUMMARY.

Lump in the throat best describes the patients' summation of a number of vague complaints in this part of the body. Vague symptoms are frequently the reflection of similar clinical conditions which may easily be overlooked.

The complaint may be present with pharyngitis, postnasal discharge, moderate cervical arthritis and esophagitis. It may occur with early retropharyngeal swelling, reaction to a foreign body lodged or passed, or a cyst in the vallecula. When it occurs with new growth or stenosis, it is an early sign. Emotional disturbances are capable of causing the complaint, and frequently affect the duration and intensity of the presenting symptoms. A number of general systemic conditions play an important part.

CONCLUSIONS.

There has been a tendency to place the patient with the complaint, lump in the throat, in the realm of the neurotic. In many cases, there is a varying element of truth in this belief.

An objective approach to the problem uncovers significant clinical findings which are the primary cause in the majority of cases. Satisfactory management of these patients embodies practice of the true art and science of medicine.

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DOES IRRADIATION OF INFANTS AND CHILDREN CAUSE CANCER OF THE THYROID?*†

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The possibility that cancer of the thyroid in children and adolescents might be related to previous irradiation of the head, neck, or thymus in infancy or early childhood, should be of great concern to otolaryngologists, the majority of whom have prescribed some form of ionizing radiation as an adjuvant in the treatment of recalcitrant ear, nose and throat diseases.

Duffy and Fitzgerald,[1] in 1950, suggested that irradiation of the thymus in infancy might be an etiological factor in cancer of the thyroid in children and adolescents. Clark,[2] in 1955, impressed with this possibility, reviewed his cases of cancer of the thyroid in this age group. He discovered in his 13 cases that all had been subjected to previous irradiation: three, to the upper chest for an enlarged thymus; three, to the neck for cervical adenitis; five, to the head and neck for enlarged tonsils and adenoids; one, to the face and anterior chest for sinusitis and peribronchitis; and one, to the anterior upper chest for pertussis.

Disturbed by this startling report of Clark's, I reviewed two cases of cancer of the thyroid in children that I had seen that year (1955) and found that they both had had irradiation for an enlarged thymus in infancy. Subsequently, I encountered two more cases, and an additional three cases were reported to me by two colleagues. The following are the case reports:

Case 1. D. R.—Female, 18 years of age. Born Feb. 1, 1940. Pertussis, Aug. 8, 1940. Chest X-ray, Sept. 5, 1940, "possibility of an enlarged

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thymus." Given 204 *r* of X-ray radiation therapy to thymus (102 *r*, Sept. 13, 1940, and 102 *r*, Nov. 29, 1940). Mass in isthmus and left lobe of thyroid noted on Nov. 10, 1951 (11¾ years of age). Surgery done on July 9, 1952 (12 years, 5 months of age). Diagnosis: Papillary carcinoma of the thyroid, with extension to the pretracheal fascia and regional lymph nodes; supplementary X-ray therapy between July 30, 1952, and Sept. 5, 1952. X-ray chest, Aug. 12, 1952, showed pulmonary metastases, which have progressively increased to the present; however, patient appears well and has no complaints. Interval between irradiation and diagnosis of cancer, 11 years, 10 months.

Case 2. S. O.—Female, 12 years of age; born June 11, 1945. Papillary carcinoma surgically removed on April 6, 1954 (8 years, 10 months of age). Supplementary X-ray therapy. Chest X-ray films were clear at that time; chest X-rays Dec., 1957, and Mar. 1, 1958, suggest metastases. Patient appears well and has no complaints.

In 1955, when I reviewed this case, the pediatrician stated that this child had had irradiation both to the thymus and nasopharynx; howver, in 1958, a record of prior irradiation could not be found; hence, there is no certainty that this patient had had prior irradiation.

Case 3. R. W.—Male, 18 years of age. Irradiation to thymus for respiratory difficulty at four months of age; mass in thyroid discovered at age 15. Surgery right radical neck dissection and total thyroidectomy for papillary adenocarcinoma. Postoperative complication: cerebral edema and partial blindness which persists; feels well.

Amount of irradiation in infancy could not be obtained, but there is no question that it was given. Interval between irradiation and diagnosis of cancer: 15 years.

Case 4. M. M.—Female, 11 years of age. Irradiation to thymus at eight months of age. Total of 800 *r* (four treatments of 200 *r* each measured in air) were given in a two-week period Jan. 5, 1948, using 200 kv. 50 cm. distance and ½ mm. of copper and 1 mm. of aluminum filtration. Total thyroidectomy and left radical neck dissection, Sept., 1952 (age 5 years, 4 months); supplemented with radioactive iodine, Oct., 1952. Diagnosis: Alveolar carcinoma of thyroid and regional lymph nodes. Present condition: feels well, but there is evidence of lung and mediastinum metastases. Interval between irradiation and diagnosis of cancer: 4 years, 9 months.

Case 5. S. B.*—Female, 19 years of age. Irradiation to thymus at six months of age. Total dose 450 *r* (150 *r*, 6/14/39, 150 *r*, 6/21/39, 150 *r*, 6/28/39); 200 kv. 1 mm. copper and 1 mm. aluminum filter. Thyroid surgically removed, 16 years of age. Diagnosis: Adenocarcinoma; patient is asymptomatic at present. No evidence of pulmonary metastases; interval between irradiation and diagnosis of cancer: 15 years.

Case 6. C. P.*—Male, 12 years of age. Irradiation of nasopharyngeal lymphoid tissue at about the age of five; dose not known. Two-stage operation for cancer of the thyroid with cervical node metastases. *1.* Right lobe of thyroid removed and right radical neck dissection; *2.* two weeks later, left lobe of thyroid removed and partial left radical neck dissection; supplementary therapy with I^{131}. At present, almost three years later, no evidence of neck recurrence or lung metastases. Interval between irradiation and diagnosis of cancer: 7 years.

Case 7. D. J.—Male, 7 years of age. Four X-ray treatments to the thymus at three months of age; exact dose not known. Surgery for

*Reported through the courtesy of Dr. William S. Freeman, Jr.

*Cases 6 and 7 reported through the courtesy of Dr. Leonard Dobson.

cancer of the thyroid with metastases to cervical nodes: total right thyroidectomy with right radical neck dissection and sub-total left thyroidectomy; supplementary therapy with I^{131}. At present, two and one-half years later, no evidence of neck recurrence or lung metastases. Interval between irradiation and diagnosis of cancer: 7 years.

REPORTS FROM THE LITERATURE.

These seven cases (one doubtful), added to those of Clark, prompted this review of the evidence for and against the possible relationship of cancer of the thyroid in children and adolescents to previous irradiation.

Duffy and Fitzgerald,[1] in 1950, reported 28 cases of histologically diagnosed thyroid carcinoma in children and adolescents, 18 years of age or younger. Ten cases in this series had been subjected to irradiation to the thymus for obstructed respiratory symptoms ascribed to "enlargement of the thymus." In all cases, these were short courses of low voltage Roentgen rays administered some time between the fourth and sixteenth month of life. These authors stated, "to propose a cause-and-effect relationship between thymic irradiation and the development of cancer would be quite unjustified on the basis of data at hand, when one considers the large number of children who have had irradiation to 'enlarged thymus'; however, the potential carcinogenic effects of irradiation are becoming increasingly apparent, and such relationships as those of thymic irradiation in early life and the subsequent development of thyroid or thymic tumors might be profitably explored." Seven years later, Duffy[3] stated, "there is strong circumstantial evidence that therapeutic X-irradiation in infants may be an etiologic factor in thyroid cancer of childhood and adolescence."

Simpson, Hempelmann and Fuller,[4] in 1955, reported a study of the subsequent history of 1,400 of 1,722 children who received X-ray therapy to the thymus gland. In 17 of these children neoplasia is known to have developed, including seven cases of leukemia and six of carcinoma of the thyroid. This is a significantly higher incidence than was found among the untreated siblings of the irradiated children or in the general population. In addition, nine treated children were found to have had thyroid adenoma. All of these cases of

cancer of the thyroid had had an amount of irradiation of 200 *r* or more.

Adding to this study, two years later Simpson and Hempelmann[5] brought the total cases traced to 1,502 treated by X-ray and 1,933 of their untreated siblings. Of those treated by X-ray the known cases of thyroid cancer increased to 11. Relative to their report of an increased incidence in leukemia, it is noteworthy that Court-Brown and Abbatt[6] reported an increased incidence of leukemia in patients with ankylosing spondilitis treated with X-ray, and Stewart[7] and associates speculated that diagnostic irradiation *in utero* may occasionally cause leukemia or cancer in the unborn child.

Winship[8,9] started a survey of childhood thyroid carcinoma in 1948, but not until 1950 did he start to inquire about a history of irradiation. In his report of 275 cases in 1955,[8] Winship stated, "Counting all the known cases in the United States of childhood carcinoma of the thyroid, almost 20 per cent have had irradiation of the thymus gland or to the neck for some other disease. This is a significant percentage."

Majarakis, Slaughter, and Cole,[11] in 1956, recorded 15 cases of cancer of the thyroid in children and adolescents between five and 20 years of age. Ten of these had a history of irradiation to the head and neck between two months and six years of age, with doses ranging from 200 *r* to 625 *r*. In this group of nine females and six males, the interval between the therapy and the diagnosis of cancer was approximately 7.8 years.

Frantz[12] told of two cases of carcinoma of the thyroid, one following irradiation for the keloid of a burn scar and the other for a giant tongue. Cancer developed after 10 years in one and 15 years in the other. The radiation in each case was in small doses.

In 1956, Fetterman[13] reported ten cases of thyroid carcinoma in children under 16 years of age. Eight of these had received prior irradiation; two for hemangiomas of the upper face and chest; two for enlarged lymph nodes; one for enlarged tonsils; and three for a presumably enlarged thymus. Fetterman emphasized the importance of repeated

questioning which doubled the number in his series known to have been previously irradiated; that is, in four of his eight cases a discovery of previous irradiation was made only after repeated questioning. Fetterman suggested that, in the series of Ravdin and Horn,[14] and of Warren and associates,[15] more pointed questioning might have elicitied information that there had been previous irradiation in a higher percentage of cases. Klopp,[16] too, felt that the incidence of previous irradiation in childhood thyroid cancer would be even higher if more correct information could be obtained by more careful questioning. He related the story of one case in which previous irradiation had been denied by the patient and the father, but later information from the divorced mother in another city revealed that the patient's thymus had been irradiated in infancy.

In an article from the Johns Hopkins Hospital, which is in the process of publication with Dr. Samuel Asper[17] as the principal investigator, the following is being stated:[27] "In a survey at the Johns Hopkins Hospital, there were found 40 thyroid carcinomas in people under 25 years of age; 17 of these had received X-ray therapy of some kind. Of the 17, six had so-called cross-fire X-ray of the tonsils; two had X-ray of the tonsils and nasopharynx; four received X-ray to the thymus; two, X-ray for acne of the upper back and face; one, X-ray for an enlarged tongue; one, X-ray for a laryngeal polyp; and one received treatment by radium pack over a hemangioma, which presented over the inner portion of the clavicle.

Notwithstanding all this evidence, there are a number of factors which have been presented in opposition to the belief that there may be an association of irradiation in infancy to carcinoma of the thyroid. In an editorial in the *British Medical Journal*,[18] referring to the reports of Rawson and associates[19] and Bielschowsky,[20] it is stated: "It could be argued, however, that the thyroid cancers in the treated children were not caused directly by the irradiation, but were in some way due to the destruction of the thymus. There is evidence (Rawson, et al.) that the thymus may be one of the sites of removal of circulating thyrotrophic hormone. Since

thyrotrophic hormone is known to play a part in the development of cancer of the thyroid (Bielchowsky), it is conceivable that the destruction of the thymus might in itself do something to provoke cancer of the thyroid by increasing the amount of thyrotrophic hormone in the blood." Counteracting this speculation is the number of thyroid cancer cases in which the previous irradiation did not involve the thymus.

It is possible, too, that some of the thyroid cancers, thought to have developed after irradiation, might have been present before the irradiation. Winship[10] pointed out that in his collected series of 400 cases of carcinoma of the thyroid in children, 12 had tumors at birth; these subsequently proved to be carcinomas. Uhlmann[21] told of a patient who failed to respond to X-ray therapy for enlarged cervical lymph nodes; two months after treatment a biopsy revealed metastatic carcinoma of the thyroid.

Uhlmann,[21] in a study of 25 patients under the age of 21 who had been referred to him for treatment of carcinoma of the thyroid during 18 years between 1938 and 1956, found that only four had had previous irradiation. During the same 18 years, he administered radiation therapy to 2,500 children for hypertrophic lymphoid tissue in the pharynx and for enlarged tonsils. In a follow-up of only 480 cases of these, not a single instance of carcinoma was found during a period of seven years. Uhlmann measured the amount of radiation that reaches the thyroid during X-ray treatment for benign hyperplasia of the nasopharynx. His customary three treatments, with a total of 375 *r* to each of two lateral fields, resulted in a maximum of 18 *r* reaching the skin above the thyroid over a period of two weeks which, according to him, is much less than the amount of radiation that reaches the thyroid during the usual fluoroscopic examination of the chest.*

Uhlmann, in his report of 1956, stated that he thought it

*A measure of fluoroscopic radiation was made by the X-ray Department of the Stanford University Medical School. The maximum dose of radiation at table-top was 3.2 **r** per minute. A skillful fluoroscopist can cone down the field of exposure to a very small area in the chest, thus avoiding the thyroid; therefore, members of the Department feel that in a well-planned fluoroscopy it would be extremely unusual to expose the thyroid to a greater amount of radiation than might be applied to the same area during therapeutic use for benign conditions.

significant that in animals cancer of the thyroid had never been produced with X-rays; however, in 1957, Doniach[22] produced thyroid cancer in seven out of 21 rats, when 1,100 rads of X-ray therapy was administered to the rat thyroid followed by prolonged methylthiouracil therapy. In only one of 13 animals with this same X-ray dose but without methylthiouracil, did thyroid cancer develop.

Uhlmann feels that, statistically, the correlation of irradiation and the development of carcinoma of the thyroid is untenable. In addition to his own cases, he refers to other reports of 68 children with cancer of the thyroid (Dailey and Lindsay, 23;[23] Horn and Ravdin, 22,[14] and Warren and associates, 23[15]) and only one had had previous X-ray therapy.

There is, on the other hand, certain experimental work referred to by Duffy,[3] which indicates that the administered irradiation is potentiated by endogenous endocrine activity in the development of thyroid cancer. Such a mechanism may account for the increased risk after the use of X-rays in the infant or growing child, compared to that in the adult. Duffy stated, "Doniach[22] has suggested that a small dose of X-irradiation may act as the initiating factor, and that the subsequent 'normal' growth and development of the thyroid from infancy to puberty may be the promoting factor in the production of the thyroid cancer in childhood. The dose of the external X-rays, or other radiation, would not then be as important as the time at which it is administered, since the mature adult thyroid tissue is apparently reasonably resistant to the development of radiation cancer."

DISCUSSION.

Bordley,[27] referring to the Johns Hopkins' survey, stated: "*1.* None of these cases received any of their ray therapy at the Johns Hopkins, which might lead one to believe that in institutions where great care was taken by people specially trained for this work, the instance of needless irradiation of surrounding tissues is greatly reduced; *2.* In the cases where X-ray was used on the tonsils and the nasopharynx, it was believed by the investigators that the coning of the X-rays

was unnecessarily large, and that the spread of the treated field was very much larger than it should have been, with pretty definite indication that the thyroid became involved in the field; *3.* There has been no evidence of any malignant changes related to irradiation with the Beta ray applicator. This . . . is perfectly natural, due to lack of penetration of the Beta rays. To my mind, it is perfectly evident that to X-ray someone because a tongue is enlarged, or because he has a laryngeal polyp, or to remove the tonsils in a child, is a rather unnecessary procedure."

Duffy[24] stated, "Although cause and effect have obviously not been proven, the possibility of radiation being a factor in the cause of thyroid and other cancers in the young is such that radiation for benign conditions in infants and early childhood should be condemned." A similar sentiment was expressed by Simpson and Hempelmann,[5] Klopp,[16] Berman,[16] Copeland,[16] Dennis,[28] and Fetterman.[13] If this be the case, what then should be the position of the otolaryngologist who has prescribed radiation as a therapeutic aid for many years? He cannot ignore the reports of the potential hazards of radiation, nor can he discount experiences such as those of the otolaryngologists of the Johns Hopkins Hospital, who have used nasopharyngeal radiation since 1924, and in whose cases it has been said,[27] there has never occurred cancer of the thyroid. A step toward the solution of this dilemma would be a re-evaluation of the indications. One might ask, "has irradiation been really necessary?" For example, could not the 2,500 cases reported by Uhlmann,[21] irradiated for enlarged tonsils and adenoids, throat infections, recurrent colds and impaired hearing, have been reduced to 250 or even 25? Does one have to resort to radiation (as reported[25]) for chronic cough, coryza, mouth breathing, snoring, nasal voice, anorexia, gagging, vomiting and choking due to postnasal drainage? Is radiation really indispensable in treating conduction deafness of tubal origin?

There is no question that much of the radiation prescribed could be dispensed with. I stated this in a previous paper,[26] and presented as an example of a certain group of patients, the case of a seven-and-a-half-year-old girl whose second

adenotonsillectomy was supplemented with X-ray irradiation. Her good hearing result (see Fig. 1) was thought at the time to be due to the combination of surgery and irradiation, but subsequent controls, as represented in Fig. 2, treated by meticulous nasopharyngeal surgery but without irradiation, produced the same good results.

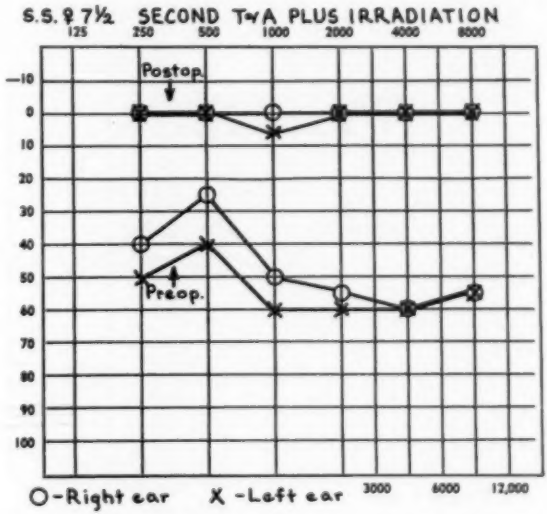

Fig. 1. Pre- and postoperative audiograms of S. S., a 7½-year-old girl. The surgery, a second adeno-tonsillectomy, was supplemented with Roentgen irradiation. This was one of a number of cases whose good results were thought, at the time, to be due to the combination of surgery and irradiation, but subsequent controls as represented by Fig. 2 suggest that adequate surgery was responsible for the improvement.

Further evidence of unnecessary use of irradiation was pointed out in the case of a 45-year-old male (see Fig. 3), who had tubal obstruction with secretory otitis conduction impairment in his only hearing ear. He had had one course of 320 *r* of X-ray irradiation to both right and left sides, another course of 700 *r* four years later, and an additional 50 mg. radium plaque, Monel-filtered, against each Eustachian

tube for a period of eight minutes. His recurrent secretory otitis and deafness did not improve. Subsequently surgical removal of the nasopharyngeal lymphoid tissue and tonsil tags, nine years after the first irradiation treatment, resulted in a cure of both the secretory otitis media and the conduction deafness. Irradiation could have been avoided had surgery

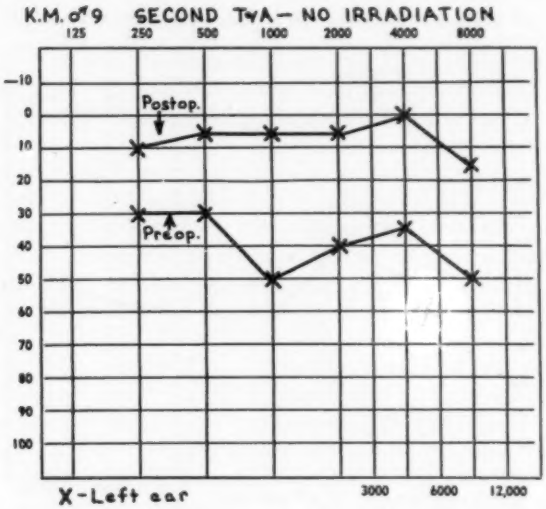

Fig. 2. Pre- and postoperative audiograms of K. M., a 9-year-old boy. His second adenotonsillectomy **without** supplementary irradiation resulted in a hearing improvement comparable to the group represented by Fig. 1 who had had supplementary irradiation.

been tried first. This also demonstrated that masses of lymphoid tissue are not always melted away by irradiation in the doses used for non-malignant conditions.

In general, it can be said that if a patient with a non-malignant ear, nose and throat disease is treated by irradiation only after a fair trial of other adequate therapy, the number of individuals who would be exposed to irradiation would be very small.

CONCLUSION.

The few cases of cancer of thyroid in children having had prior irradiation, herein presented, in addition to those previously reported, do not permit one to conclude that irradiation of infants and children causes cancer of the thyroid to appear in later years; however, the possibility is sufficiently

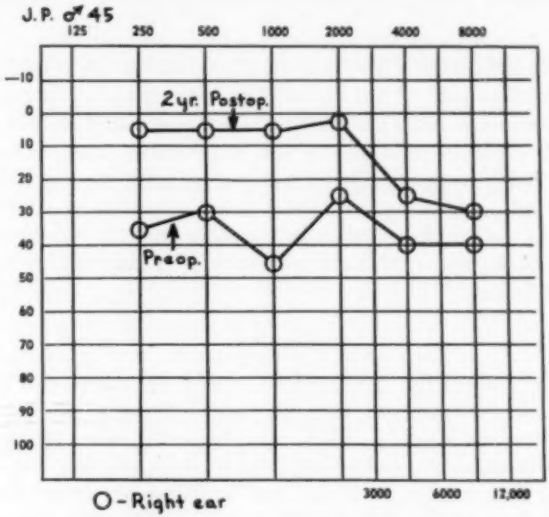

Fig. 3. Pre- and postoperative audiograms of Mr. J. P., age 45. Chronic serous otitis media in right ear. Course of X-ray irradiation to nasopharynx in May, 1947, and October, 1951, and one treatment with radium plaque, Monel-filtered, in December, 1951. No improvement until nasopharyngeal lymphoid tissue and tonsil remnants were removed surgically in June, 1956.

startling to make one suspicious of potential hazards of even small doses of radiation in children. Until the evidence is conclusive, for or against this relationship, one should use great restraint in undertaking irradiation for any nonmalignant condition in infants and children. For the otolaryngologist, this caution should prove no handicap, for there are very few benign ear, nose or throat diseases of

infants and children which absolutely require irradiation therapy.

SUMMARY.

1. Seven cases of thyroid cancer in children are reported; six of them are known to have had therapeutic irradiation in infancy. These cases add further weight to the many similar reported cases of childhood thyroid cancer, pointing to X-ray as a major etiologic factor for thyroid cancer in young persons.

2. The evidence for the relationship of irradiation and childhood cancer of the thyroid is inconclusive. Some series reported show little correlation; however, in the light of reported evidence it appears not improbable that irradiation of the head, neck or mediastinum in infancy may increase the chance of getting thyroid cancer some years later. Unless the etiologic connection comes to be disproved, it would appear wise not to use radiation on children if any other therapy can be found.

ADDENDUM.

Since presenting this subject before the American Laryngological, Rhinological and Otological Society, Inc., at San Francisco on May 21, 1958, an additional case appeared at the Stanford University Hospital; it was called to my attention by Dr. Joseph P. Kriss, and is being presented through the courtesy of Dr. Carleton Mathewson, Jr., who is in charge of the patient.

Case 8. R. R., 9 years of age. Irradiation to thymus at three weeks of age. Total of 225 *r* were given in a three-week period starting April 18, 1949, using 140 kv. peak, 20 milliamperes, filtration ¼ mm. copper and 1 mm. aluminum, half value layer .42 mm. of copper, target skin distance 50 cm., cone field size 10x10 cm., but irradiated size 6x10 cm. In June, 1957, surgery, consisting of a left radical neck dissection with the removal of the left lobe and isthmus of the thyroid, was done. On November 4, 1957, additional surgery, which involved a right radical neck dissection and the removal of the right lobe of the thyroid, was done. In January, 1958, the child developed dyspnea. A chest X-ray revealed diffuse, bilateral pulmonary metastases. Additional therapy in the form of I^{131} was given in June, 1958.

Interval between irradiation and diagnosis of cancer: eight years.

Average interval between irradiation and diagnosis of thyroid cancer for all cases herein reported: 9.8 years.

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ALUMNI ASSOCIATION OF THE NEW YORK EYE AND EAR INFIRMARY.

The Annual Spring Meeting of the Alumni Association of the New York Eye and Ear Infirmary last April was so well received that it has been decided to expand next year's meeting, which will take place from April 20-23, 1959.

Symposia and lectures on Hearing Rehabilitation, Endoscopy, and Ear Surgery will be conducted. It is also planned to offer refresher courses in Mastoid and Fenestration Surgery and Stapes Mobilization Techniques.

More complete information regarding the meeting will appear in a later issue of THE LARYNGOSCOPE.

CYTOLOGIC DIAGNOSIS AND PROGNOSIS IN CARCINOMA OF THE MOUTH, PHARYNX AND NASOPHARYNX.

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According to a survey reported in the *Cancer Bulletin*,[1] mouth and lip cancer comprise 10 per cent of all male cancer. This same publication estimates that as high as 80 per cent of deaths from cancer of the mouth could be prevented by early recognition and prompt adequate treatment.

No attempt will be made in this paper to discuss clinical findings or treatment in cancer of the mouth and nasopharynx. The sole purpose of this study is to describe a valuable adjunct to the early diagnosis of cancer of these areas, and to demonstrate a means of evaluating radiation treated lesions in which repeated biopsies would prove harmful.

It was in 1949 that the late Dr. Morrison,[2] Dr. Wu and I first published the results of the application of the Papanicolaou-Traut[3] smear technique of cytologic diagnosis to cancer of the upper respiratory tract. In that first publication we were primarily concerned with the application of the smear technique to the early diagnosis of lesions in the nasopharynx. The success achieved in this area soon led to the application of the same technique to the early diagnosis of lesions in the mouth, pharynx and even larynx. The results of the cytological studies made in these areas have been excellent. It should again be emphasized, as it was in the original paper, that this procedure is not intended to replace tissue biopsy and histological examination.

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Technique: Various methods have been described for taking smears in the mouth and nasopharynx. Gladstone[3] used both gelfoam and cellulose sponges. The sponge was rubbed over the surface of the lesion and then sectioned. Thirteen mouth lesions were thus smeared. Two of these had a positive tissue biopsy for carcinoma and these two also had positive smears. The remainder were all negative. Montgomery[5] scraped the mucosa with a Woodson's No. 2 metal plastic instrument, and Miller[6] and his co-workers used a wooden spatula. Both obtained good smears; however, our original technique, slightly modified, still seems the simplest. A tightly wound cotton applicator is rubbed over the surface of the lesion and the adjacent area. The rubbing must be firm but not sufficient to produce active bleeding. The cotton applicator is then rubbed on the surface of a clean, dry glass slide. The slide is immediately immersed in the standard fixative solution of equal parts of 95 per cent alcohol and ethyl ether. The slide is kept in the solution until ready to be stained. Slides may be left in the fixing solution indefinitely or stained after a minimum of one-half hour.

The staining technique follows the modified method of Papanicolaou and Traut[7] with but one exception. EA 50 has been used to replace EA 36 or 25 of step No. 6. More recently the technique of Pharr, Wood and Traut[8] has been followed in the staining procedure.

Normal Cytology: A study of the normal cytology of the respective areas being smeared is essential to the diagnosis of the malignant smear. In general, the histological characteristics of the area determine the cells found in the normal smear. In the nasopharyngeal smear there may be found ciliated columnar cells, columnar cells which have lost their cilia, lymphocytes and polymorphonuclear leukocytes. Occasionally and particularly in infection histiocytes, epithelial cells, bacteria and erythrocytes are seen.

In the mouth and pharynx ciliated cells are uncommon, and squamous epithelial cells predominate. The staining of these cells varies from blue to red to yellow. While the determining factors in the color range appear to be age or maturation of

the cell and degree of cornification, the staining color is not specific. As might be expected, greater numbers of cornified cells appear in the tongue and gingival smears; fewer in cheek and post-tonsillar smears.

Fifty normal smears from adults of almost equal sex distribution and varying ages show no variation of cytology for sex, age or phase of the menstrual cycle. Such studies were reported by Miller et al.[6] and Montgomery[5] in 1951.

In the normal smear much nuclear variation is encountered both as to size and shape and position in the cell, but the chromatin content is usually evenly distributed and the cell cytoplasm has a distinct, clearly defined border. The larger cells in mouth smears range from about 70 to 100 mμ in size and the medium-sized cells range from about 35 to 40 mμ. The nucleus is small compared to the size of the cell, being about 10 mμ in the large cells and 5 mμ in the medium-sized cells.

Pre-Malignant Cytology: Smears in ten cases of leukoplakia of the mouth were examined. As noted by Weinman[9] and Montgomery and von Hamm[10] there appears to be no characteristic cell type.

Malignant Cytology: Mouth smears from malignant areas are usually rich in cells; the cell nucleus and nucleolus are enlarged; multiple nucleoli are frequent, and the diagnosis of malignancy may be made on the basis of thickened nuclear membranes, hyperchromasia, abnormal clumping of chromatin material and uneven distribution of chromatin bits. The cell itself has ill-defined borders; may be distorted or even without a visible, true wall. Clumps of these cells are characteristic, but they may be found singly. Of course, the normal cytology of squamous cells, leukocytes and clasmatocytes, is also found in the malignant smear.

Post-Radiation Cytology: In carcinoma of the mouth and nasopharynx which is under treatment with X-ray, malignant cells usually disappear or show senile changes after about the third or fourth week of therapy. There is an increase in polymorphonuclear leukocytes and in pink or red staining

squamous cells. This is especially noticeable in areas of ciliated epithelium where ciliated cells tend practically to disappear. The staining characteristics of the cells are probably due to increased keratinization and decreased production of immature cells, with consequent increase in aged or damaged cells.

RESULTS.

One-hundred-and-four cases of benign lesions of the nasopharynx, mouth, tonsil, and larynx had Papanicolaou smears made without a false positive for carcinoma. Peters[11] in a very recent report smeared 401 cases of leukoplakia, sub-

TABLE I.

	No. of Cases of Carcinoma	Positive Smears	Negative Smears
Nasopharynx	12	11	1
Mouth	25	24	1
Tonsil	14	14	0
Larynx	12	11	1

mucous fibrosis, scleroma and other benign conditions in the mouth and obtained three false positive smears for a total error of 0.7 per cent. Silverman[12] and his co-workers at the University of California, in a fine study soon to be published, reported no false positive mouth smears in a group of 60 patients.

Table I records the results of the cytological studies in the nasopharynx, mouth, tonsil and larynx.

Three cases of carcinoma of the mouth were studied cytologically during and after radiation treatment. All smears were negative by the fourth week of therapy. One remained negative and the patient showed no evidence of recurrence five years post-radiation. The other two (anterior pillar and tonsil) yielded positive smears within two months after therapy and were confirmed by subsequent biopsy. Silverman[12] and his co-workers have reported a case which illustrates the point very well. Their patient was a 46-year-old male found on examination and biopsy to have a lympho-

TABLE II.

RESULTS OF CYTOLOGICAL STUDIES IN CARCINOMA OF THE MOUTH.

	Montgomery[12]	Wahl[11]	Peters[13]	Morrison[2]	Peters[11]	Silverman[12]	Hopp	Totals
Number of Patients	15	41	114	10	194	19	25	418
Positive Smears	13	41	110*	10	186**	18	24	402
Negative Smears	2	0	4	0	8	1	1	16
Positive Biopsies	15	41	114	10	162	18	25	385
Negative Biopsies	0	0	0	0	23	1	0	24

* 8 smears reported suspicious grouped with the positive.
**32 smears reported suspicious grouped with the positive.

epithelioma of the soft palate. The lesion disappeared after X-ray therapy, but a small ulceration developed a few months later. The problem was to differentiate between recurrence and post-radiation necrosis. Biopsy in the irradiated area could have produced considerable difficulty if the lesion proved benign. The initial smear was inconclusive but suspicious. Repeat smears yielded a positive for malignancy. This was then confirmed by biopsy.

In Table II the statistics of several mouth smear studies have been assembled. Two cases of carcinoma of the mouth were first discovered on routine smear by Peters.[11] One had been thought to have an ulcerative stomatitis and the other an hypertrophy of the posterior pillar. In Silverman's[12] series, four cases of mouth lesions were first diagnosed as malignant by cytologic studies.

In conclusion, the smear technique is not intended to replace tissue biopsy. It is a simple method of diagnosis, however, and can enable one to diagnose malignancy at an earlier stage. In addition, radiation treated lesions can be kept under long term observation. As Peters[11] so well stated: "Repeated biopsies might be difficult, but repeated smears and occasional biopsy are possible and often desirable. The reliability of the cytologic interpretation increases with the experience of the interpretor. Cytology does not replace histology but supplements it."

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The University of Illinois College of Medicine Department of Otolaryngology announces its Annual Otolaryngologic Assembly from September 29 through October 5, 1958. The Assembly will consist of an intensive series of lectures and panels concerning advancements in otolaryngology, and evening sessions devoted to surgical anatomy of the head and neck and histopathology of the ear, nose and throat. Interested physicians should write direct to the Department of Otolaryngology, 1853 West Polk Street, Chicago 12, Ill.

SOFT TISSUE ROENTGENOGRAPHY OF THE NASOPHARYNX FOR ADENOIDS.*†

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INTRODUCTION.

The accurate evaluation of the adenoids is of great importance in the proper management of naso-sino-nasopharyngeal problems so frequently encountered in infants and children. Both the otolaryngologist and pediatrician appreciate that the clinical examination to determine the status of the adenoids is often difficult and unsatisfactory in these young patients. Posterior rhinoscopy is usually impossible, with the result that digital palpation of the nasopharynx is the clinical method most often employed to estimate the degree of obstruction that exists in the nasopharynx; however, this diagnostic procedure is crude and fraught with serious difficulties. It is questionable whether a probing finger can reach the vault of the nasopharynx behind the vomer, or determine the amount or depth of lymphoid tissue while an unwilling patient is crying and struggling. Thus, information obtained in this manner is often incomplete and even inaccurate. In addition, the psychic trauma which may accompany this manipulation is undesirable.

In contrast, we have found that radiographic examination of the nasopharynx is a simple procedure which can be performed satisfactorily and with ease in children of all ages. Roentgen ray exposure is minimal, approximately that of a routine chest examination. We have used this method in more

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than 150 cases of naso-sino-nasopharyngeal problems, both pre- and post-adenoidectomy, as part of a thorough investigation. This also included X-ray examination of the sinuses, bacteriological and cytological studies of nasal and sinus secretions and allergic tests. Our experiences have revealed that the information obtained by radiography of the nasopharynx is usually more complete and accurate than that obtained by other methods, and can be related to the surgical scope when operation is indicated. In addition, we have noted certain aspects in the radiographic evaluation of the nasopharynx which have not been previously discussed in the literature. We are reporting these data and experiences to emphasize the value of radiographic examination of adenoids.

REVIEW OF LITERATURE.

Although the literature contains relatively little concerning the radiological investigation of the nasopharynx, nevertheless the potential value of this subject has been appreciated by a number of authors. The majority of these, as might well be expected, have been radiologists. In 1898, shortly after the clinical value of X-rays became known, Mignon[1] mentioned radiographing the adenoids in his thesis on "Radiology of the Respiratory Tract." In 1904, Scheier[2] reported the demonstration of the epipharynx and enlarged adenoids roentgenographically and the disappearance of the adenoids from the X-ray plate postoperatively. Technical progress, however, had not advanced sufficiently as yet to permit satisfactory results routinely, and little more was written on this subject for the next 20 years. In 1925, Grandy[3] described the soft tissue opacity of the adenoids in the nasopharynx and the narrowing of the airway locally. He also noted the increased airway following adenoidectomy.

Bernfeld[4] in 1927 was the first to stress the importance of the size of the nasopharynx in relation to the problem of obstruction of the airway by adenoids, the difficulty of their complete removal and the frequency of recurrences. He believed that complete resection was more difficult and recurrences of much greater frequency in patients with small epipharyngeal cavities. In 1929, Schüller[5] discussed the gen-

eral subject of bony deformities of the nasopharynx as demonstrated roentgenographically. He described the variations in size and appearance of this airspace, and quoted Testut and Jacob who believed that the infantile nasopharynx was comparatively small with a flat vault, while the adult nasopharynx was larger with a high angular or rounded vault. It was also noted that tumors of the epipharynx, including adenoids, caused more obstruction if the cavity was small and narrow. Moulinard[6,7] in 1949 also believed that the infant type of nasopharynx was relatively small with an axis parallel to the base of the skull, while the adult epipharynx was larger with a more vertical antero-posterior axis. Other reports[3,8,9,10,11,12,13] similarly indicating the value of the radiographic appearance of enlarged adenoids have appeared sporadically in the literature. Brief descriptions of the method and its merits have been published in standard reference texts.[14,15]

TECHNIQUE.

We have observed that a lateral view of the nasopharynx at a six foot target-film-distance and with factors permitting rapid exposure (1/30 to 1/60 second) to avoid motion blurring is most satisfactory. A non-Bucky technique is preferred, since the gradations on the film are more gradual, the contrasts less and the soft tissues are shown more completely. We have used tomographic techniques in the profile view of the nasopharynx, as have others,[16] but have found that this procedure does not yield additional information. Lipiodal studies[17] have not proved of value.

Radiographic visualization of the adenoids results from the contrast between the water-density of the adenoidal soft tissue mass and the radiolucency of the surrounding nasopharyngeal airway. It is, therefore, apparent that the larger the nasopharyngeal airway, the more likely are margins of the adenoids to be demonstrated, even if enlarged. The anterior, superior and posterior walls of the nasopharynx are bony and immobile. The floor of the nasopharynx is the superior surface of the soft palate and mobile. Depression of the soft palate increases the nasopharyngeal airspace and improves the visibility of the adenoids. Elevation of the

soft palate often brings this soft tissue density in contact with the soft tissues of the posterior pharyngeal wall, and thus reduces both the airway and the demonstrability of the adenoids. We have shown, as have others,[10,13,15,18] that the soft palate is in a mid-position with the nasopharynx open during the simple suspension of respiration. Phonation, swallowing and crying elevate the soft palate and diminish the nasopharyngeal airway. Inspiration and expiration of air through the nose depresses the soft palate about equally and thereby increase the nasopharyngeal airway. Position of the patient, whether supine, lateral recumbent, sitting or standing, plays no major role in elevation or depression of the soft palate; however, there is less elevation of the shoulders and thickening of the cervical soft tissues when the patient's head is held erect and the neck is slightly extended. For these reasons our lateral views of the nasopharynx are made with the examinee erect; *1.* with respiration suspended, and *2.* during nasal inspiration.

ROENTGEN ANATOMY.

The nasopharynx is that portion of the respiratory airway situated behind the choanae and above the soft palate.[5,19] The roof of the nasopharynx is formed by the basisphenoid and basiocciput, the posterior wall by the condyloid portion of the occipital bone and the adjacent anterior arch of the atlas, the floor by the superior surface of the soft palate, and the anterior boundary by the vomer and choanae medially, and the pterygoid plates laterally (see Fig. 1). The roof, posterior wall and floor of the space are easily recognized on the radiograph. The choanae and vomer, of considerable importance in the evaluation of adenoidal obstruction, cannot be directly identified on the film; however, an accurate estimation of the location of the choanae may be obtained since they are on the same plane as the posterior edge of the vomer. The lower edge of this bone begins at the posterior margin of the hard palate (the palate bone) and extends upward and backward to articulate with the rostrum of the sphenoid at the under surface of the base of the skull. The plane of the posterior margin of the hard palate can be delineated clearly on the film. From this point a line drawn upward and backward to

the point where the upper anterior margin of the pterygoid plate joins the body of the sphenoid corresponds closely to the posterior edge of the vomer and the position of the choanae (see Fig. 2). This line lies very near the posterior margin of the antrum, and the latter also may be utilized as an adequately accurate position of the choanae.

The two determinants in nasopharyngeal obstruction are

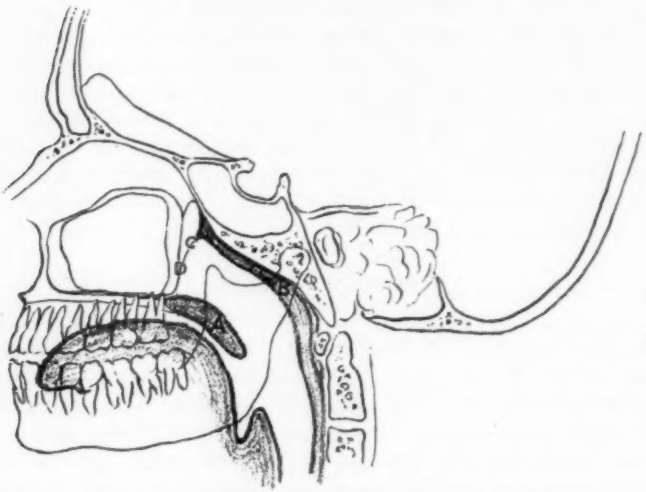

Fig. 1. Diagram of anatomy of nasopharynx. Nasopharynx located between soft palate (A) and mucous membrane of roof (B). The line of the posterior edge of the vomer is approximated by either the anterior edge of the pterygoid plate (C) or the posterior wall of the maxillary antrum (D).

the degree of adenoidal enlargement and the size and shape of the nasopharyngeal space. We believe that the importance of the latter has not been sufficiently recognized. Both the size and shape of the nasopharyngeal space vary considerably. The major factor in this variation is the shape of the roof or fornix of this cavity. Usually, the roof is highest anteriorly, just behind the choanae, where the vomer joins with the inferior surface of the body of the sphenoid bone (see Fig. 2). From this point the fornix slopes downward and backward at

an angle of about 30° with the horizontal as it extends towards its junction with the posterior wall of the nasopharynx. It is thus apparent that the largest nasopharyngeal airway is usually the anterior region just behind the choanae.

The significance of the anatomical arrangement just described is further emphasized by the effects of the action of

Fig. 2. X-ray of dried skull. Posterior margin of vomer marked with opaque paste. Note close relationship to anterior margin of pterygoid plate and posterior wall of maxillary antrum.

the soft palate. The size of the posterior part of the epipharynx can be increased by the depression of the soft palate which occurs during inspiration or expiration. The movement of the soft palate, however, is least at its attachment to the hard palate just behind the choanae. As a result, the available airway just behind the choanae is least affected by the soft palate motion. Because of the lack of soft palate motion in this plane, the high vault of the nasopharynx anteriorly can be considered a compensatory arrangement to furnish the largest possible nasopharyngeal airway for respiration.

Patients have been encountered, however, in whom the arch of the roof anteriorly is comparatively flat rather than high, and the entire epipharyngeal vault is low (see Fig. 3). The supero-inferior diameter of the nasopharynx is thus diminished and the entire nasopharyngeal airway decreased. This may be partly compensated posteriorly by marked depression of the soft palate, but anteriorly behind the choanae such lowering of the soft palate is not possible. The critical plane for nasopharyngeal obstruction is, therefore, to be found anteriorly just behind the choanae in patients with low,

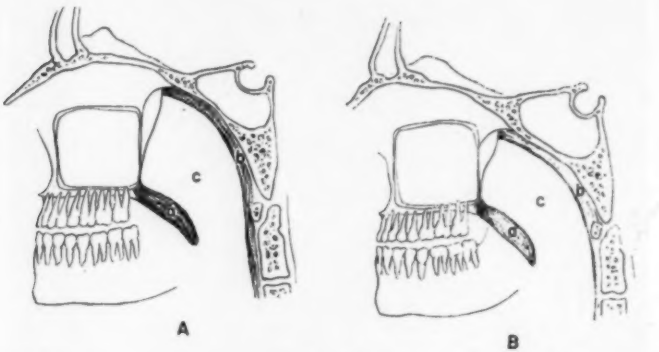

Fig. 3. Diagram of nasopharynx with high vault (A) and low vault (B). Soft palate (a), mucous membrane roof of vault (b), nasopharyngeal space (c).

poorly arched nasopharyngeal vaults. Clinically, the anterior "critical region" of the nasopharynx is the zone most difficult to explore by digital examination. Thus, adenoidal tissue in this recess might not be discovered by this diagnostic method.

We have observed that usually the nasopharyngeal areas in young children are smaller than in adults, and the vault is lower and less well arched; however, considerable variations in size and height of the vault existed both in the children examined and in the adult radiographs studied for comparison. This individual variation plays an important role in the matter of airway obstruction. In some patients moderate enlargements of the adenoids caused complete obstruction of com-

paratively small nasopharyngeal airways, while in other instances even larger adenoids did not completely occlude comparatively larger nasopharyngeal spaces.

ROENTGEN EXAMINATION OF ADENOIDS.

The mucous membrane lining the roof and adjacent posterior wall of the pharynx is cast in folds and diffusely infiltrated with lymphoid tissue throughout its submucosa. In areas the lymphoid tissue is relatively more abundant, and forms conglomerate nodular masses with crypt-like invaginations of the surface epithelium. These are the pharyngeal tonsils or adenoids. When infection occurs, there is swelling and enlargement of the diffusely dispersed lymphoid tissue and the nodular lymphoid masses, which result in thickening of the mucous membrane lining of the nasopharyngeal vault. The increased width of the mucous membrane may be essentially flat if the diffusely dispersed lymphoid tissue is predominantly involved; nodular in shape if the coalescent lymphoid masses are mainly affected, or a combination of both types. Usually the combined form is encountered with marked adenoidal enlargements. Nodular enlargements of lymphoid tissue may occur anywhere in the mucosa of the roof. The adenoids develop rapidly after birth, attain their maximum size in early childhood and begin to involute at about 8 to 10 years. In most instances regression is nearly complete by 12 to 14 years of age.

Radiographically, the normal mucous membrane lining of the nasopharyngeal roof is seen as a thin band of soft tissue density lying immediately beneath the inferior surface of the body of the sphenoid bone. It begins antero-superiorly in the fornix just behind the choanae and extends backward to merge with the mucous membrane lining of the posterior pharyngeal wall. The free surface of this mucosal band can be visualized on the roentgenogram, because it borders the air-containing nasopharyngeal cavity (see Fig. 4). It measures no more than 6 mm. in width in the normal nasopharynx, and runs parallel with the surface of the sphenoid bone which it covers. The normal mucous membrane shows no nodularity. A well defined, patent, radiolucent nasopharyngeal airway is

clearly delineated between the free edge of the mucous membrane of the roof and the superior surface of the soft palate which forms the floor of the nasopharynx.

As the adenoids enlarge, the width of the soft tissue band under the sphenoid bone increases, resulting in a corresponding narrowing of the adjacent radiolucent airway. In addition to the general increase of width, local areas of greater

Fig. 4. Normal nasopharynx.

nodular densities may be seen at the sites of proliferation of conglomerate lymphoid masses which further narrow the airway locally. The adenoids are classified as moderately enlarged when the width of the mucosal soft tissue band is greater than 6 mm. and there is mild superimposed nodulation, but nasopharyngeal airway is considered adequate (see Figs. 5-A—5-B). Marked enlargement is shown by a further increase in width of the soft tissues under the sphenoid bone, usually associated with local crescentic swellings of the mucosa. The nasopharyngeal airway is markedly narrowed but not completely occluded (see Figs. 6-A—6-B). The final stage of enlargement is characterized by complete obstruction of the airway by greatly widened and nodular mucous mem-

Fig. 5-A. Moderately enlarged adenoids. Respiration suspended.

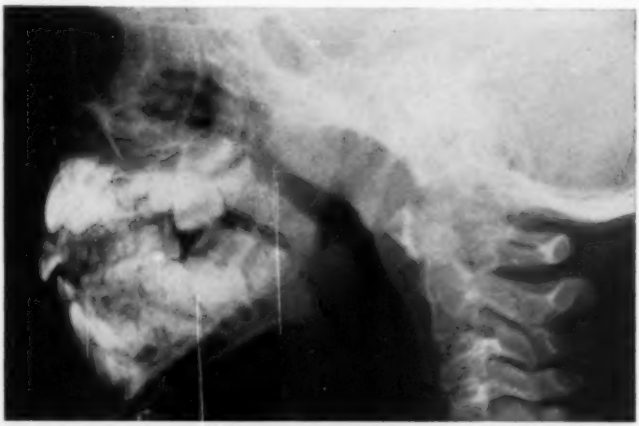

Fig. 5-B. Moderately enlarged adenoids. During nasal inspiration.

Fig. 6-A. Markedly enlarged adenoids. Respiration suspended.

Fig. 6-B. Markedly enlarged adenoids. During nasal inspiration.

brane, which extends downward from the sphenoid bone and comes in contact with the superior surface of the soft palate (see Figs. 7-A—7-B).

The importance of the mobility of the soft palate floor of the nasopharynx becomes evident in the study of cases of enlarged adenoids. In each instance films are made during "respiration suspended" and during "nasal inspiration." When the major adenoidal enlargement is in the posterior part of the roof, the nasopharyngeal airway may appear occluded during suspension of respiration as the adenoids lie in contact with the superior surface of the posterior portion of the soft palate; however, on the "nasal inspiration" film the soft palate is depressed so that a distinct, although narrow airway is demonstrated (see Figs. 5-B—6-B), and some nasal respiration is permitted. If the adenoids are sufficiently enlarged, the airway is completely blocked at all times (see Fig. 7-B). If the major adenoidal enlargement, usually nodular, is situated anteriorly in the nasopharynx just behind the choanae, the degree of obstruction cannot be alleviated by the downward motion of the soft palate since this motion is minimal at this site (see Fig. 6-B). For this reason, adenoidal obstructions of the nasopharynx anteriorly are not significantly affected by nasal respiration.

Radiographic differential diagnosis of enlarged adenoids is usually not difficult. A widened, nodular soft tissue mass attached to the roof of the nasopharynx associated with narrowing of the nasopharyngeal airway almost invariably signifies adenoidal enlargement in young children. A common source of error is the shadow of the earlobe projected into the nasopharyngeal airway. Uusually, this shadow lies just below and slightly anterior to the external auditory canal (see Fig. 8-A); however, if the child's head is not in a true lateral position during the filming, the earlobe may be projected into the anterior portion of the nasopharyngeal airway and cause confusion (see Fig. 8-B). Identifying the earlobes in each case should eliminate this problem. If doubt still exists, the earlobes may be folded up out of the area and fixed to the temples by adhesive tape prior to the filming.[14]

Choanal and nasopharyngeal polyps, fibromas and malignant neoplasms may be observed as soft tissue masses in the nasopharynx. The choanal polyps extend backward from the choanae into the anterior portion of the nasopharynx and do not arise from the vault. On radiographs these soft tissue circumscribed densities may be seen separated from the mucous membrane of the vault by a thin band of nasopharyngeal airway (see Fig. 9). In addition, the polyps, fibromas and malignant tumors occupying the nasopharynx usually appear after the age when the adenoids have involuted. The juvenile and adolescent fibromas as a rule form huge, homogeneously dense, round and sharply circumscribed opacities quite different from the ovoid, lobulated masses seen attached to the vault in the younger children.

Radiography of the nasopharynx is a useful method of assessing the success of adenoidectomy procedure. Following surgery the entire thickened, lobular soft tissue density observed in the nasopharynx preoperatively is seen to have disappeared. The mucosa of the roof is noted as a narrow, smooth band of soft tissue density without nodulation, and the nasopharyngeal airway is wide and patent (see Figs. 10-A—10-B). Any areas of residual adenoids are easily identified as local soft tissue nodules projecting into the airway. Similarly, recurrent adenoidal enlargement following surgery is apparent as band-like or nodular thickening of the mucous membrane of the vault which projects into the nasopharynx and narrows the airway exactly as do the primary adenoidal enlargements which have not been subjected to operation.

In this series of cases, 46 children were evaluated for complaints lasting from several months to two years after tonsillectomy and adenoidectomy. In 26 instances enlargement of adenoid tiisue, usually high in the vault, could be demonstrated by roentgenography.

We have indicated that the anterior area of the vault of the nasopharynx is the "critical region" physiologically. This term can be applied also to the surgical considerations of this area. Study of the anatomy of the vault of the nasopharynx by roentgenography reveals the reasons for the contentions

Fig. 7-A. Complete obstruction of nasopharynx by greatly enlarged adenoids. Respiration suspended.

Fig. 7-B. Complete obstruction of nasopharynx by greatly enlarged adenoids. During nasal inspiration.

Fig. 8-A. Usual position of earlobe, anterior and inferior to external auditory meatus.

Fig. 8-B. Earlobe more anteriorly located in nasopharyngeal space due to slight rotation of head.

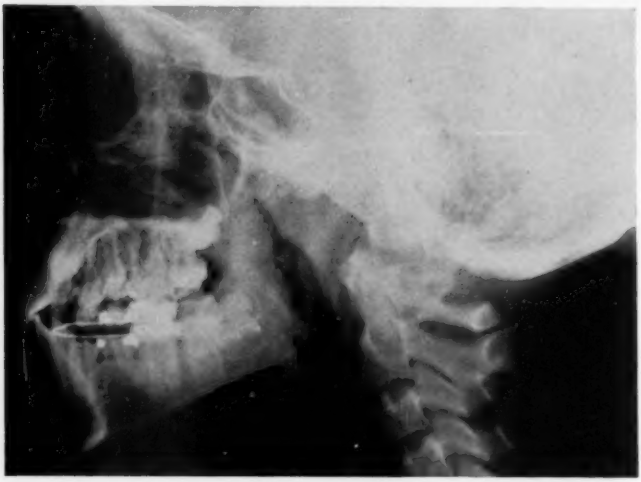

Fig. 9. Choanal polyp in nasopharynx.

Fig. 10-A. Complete obstruction of nasopharynx by adenoids preoperatively.

Fig. 10-B. Clear nasopharynx postoperatively.

Fig. 11-A. Case 2. Enlarged adenoids with respiration suspended prior to third operation.

Fig. 11-B. Case 2. Enlarged adenoids during nasal inspiration.

that complete and direct exposure of the vault of the nasopharynx is necessary to accomplish a thorough adenoidectomy. Yankauer[20] has devised a speculum; Love,[21] Baron,[22] Lothrop,[23] Senturia[24] and others have offered palatal elevators for this purpose. Meltzer[25] has shown the need of using punch forceps under direct vision to remove remnants of lymphoid

Fig. 12-A. Case 4. Normal nasopharynx.

tissue in the fossa of Rosenmueller and high in the vault. Pedersen[26] suggests the intranasal route for removal of the adenoid tissue in the area we have called the "critical region" of the vault. Thus, by demonstrating the anatomy and physiology, roentgenography can be helpful in the planning of the surgical approach of a complete adenoidectomy, in addition to its diagnostic value.

Fig. 12-B. Case 4. Markedly cloudy maxillary antrums.

CLINICAL APPLICATIONS—CASE HISTORIES.

The main possibilities in the differential diagnosis of naso-sino-nasopharyngeal problems in children which confront the otolaryngologist include the following condition: *1.* Adenoidal enlargement with varying obstruction and/or disease; *2.* nasal infection and/or sinus infection; *3.* vasomotor rhinitis and sinusitis; *4.* related ear conditions; *5.* occasional polyps and neoplasms. For satisfactory solution of these problems, often a complete work-up is required; this must include a detailed history and careful examination, radiographic studies of the nasopharynx and the paranasal sinuses, spreads and cultures of the nasal and sinus secretions, as well as allergic investigations when the latter appear indicated. The radio-

graphic studies, while of considerable importance, constitute but one aspect of the complete investigation. Often, all the elements of the work-up interlock to form a pattern which indicates the abnormality as well as the therapeutic approach. A number of brief case reports will serve as illustrations.

PRIMARY ADENOIDAL ENLARGEMENT WITH OBSTRUCTION.

Case 1. C. W., age 8, complained of constant mouth breathing, nasal obstruction and nasal speech. Examination revealed no abnormal findings in nose, sinuses or ears, except small amount of secretion in nasal cavities. Tonsils were moderately enlarged. X-rays of nasopharynx showed greatly enlarged lobular adenoids filling the nasopharynx and causing obstruction during "respiration suspended" and almost complete occlusion during "nasal inspiration." Culture of nasal secretions yielded large growths of micrococcus catarrhalis and diphtheroids, a bacteriologic finding often seen in association with active adenoid tissue. At operation the enlarged adenoids were removed, following which symptoms disappeared. Postoperative X-rays of the nasopharynx showed a clear, widely patent airway.

POSTOPERATIVE ADENOIDAL ENLARGEMENT, EAR INFECTION, IMPAIRED HEARING.

Case 2. C. F., age 7, had tonsillectomy and adenoidectomy performed at age of two years. For the following five years patient had repeated infections of the ears and upper respiratory tract. Hearing became impaired. A second adenoidectomy was performed eight months prior to his first visit, but hearing did not improve. Examination revealed no evidence of nasal or sinus disease. The tympanic membranes were congested and retracted. Audiogram indicated a moderate conductive hearing impairment. X-rays of the nasopharynx showed enlarged adenoids high in the vault (see Figs. 11-A—11-B). These enlarged adenoids were removed at a third adenoidectomy. Following this operation, the tympanic membranes assumed a normal appearance, and the hearing returned to a normal level.

IMPAIRED HEARING UNRELATED TO NASOPHARYNGEAL OBSTRUCTION.

Case 3. L. G., age 8, had impaired hearing. A tonsillectomy and adenoidectomy had been performed previously, and an evaluation of the hearing loss in relation to adenoidal disease was requested. Examination revealed no evidence of nasal or sinus disease. Tympanic membranes were normal. Radiographic studies of the nasopharynx showed no evidence of adenoids. Direct nasopharyngoscopy revealed no lymphoid tissue. Audiometric study revealed the hearing impairment to be of the perceptive type.

NASAL SYMPTOMS, NASAL DISEASE; NO ADENOIDAL ENLARGEMENT.

Case 4. J. B., age 3, complained of difficult nasal breathing, night snorting, recurrent episodes of fever. Examination revealed congested turbinates and a moderate amount of thin secretion in both nasal cavities. Radiographic investigation showed no adenoidal enlargement in the

nasopharynx but marked clouding of the maxillary antrums (see Figs. 12-A—12-B). Spreads of the nasal secretions revealed numerous cocci, and cultures yielded large growth of Staphylococcus aureus A. Sensitivity tests showed that this staphylococcus was sensitive only to achromycin, terramycin and chloromycetin. Systemic and local tetracyclin therapy was initiated and the condition cleared rapidly.

NASOPHARYNGEAL OBSTRUCTION DUE TO POLYP.

Case 5. S. T., age 11, complained of difficulty in breathing particularly through the left nostril, mouth breathing and nasal speech. An allergic history was elicited. Examination revealed a large nasopharyngeal polyp obstructing the left choana. X-rays of the nasopharynx demonstrated the polyp arising from the region of the choanae and projecting into the anterior portion of the nasopharynx. It was seen to be distinct from slightly enlarged, non-obstructive adenoids in the vault (see Fig. 9). Breathing difficulties disappeared after removal of the polyp.

CONCLUSIONS.

Radiographic examination of the nasopharynx is an accurate, simple and safe method for determining the size, shape and position of enlarged adenoids, and the degree of nasopharyngeal obstruction they cause. For this reason it is one of the important procedures in the total investigation of naso-sino-nasopharyngeal problems in children. Review of the literature suggests that this diagnostic method is utilized less frequently than it deserves. It should replace the crude and inadequate method of digital palpation. The anatomy of the nasopharynx with emphasis on variations in its size and shape, especially with reference to diagnostic and surgical problems, is presented. The radiographic appearance of adenoidal enlargement is described. Differential diagnosis is discussed and illustrative case reports are recorded.

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A CLINICAL LOOK AT PROGRESS IN EVALUATION OF HEARING FUNCTION.*

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INTRODUCTION.

Progress in the development of hearing tests has followed a clinical need on the part of the otologist for more information which would contribute to a more complete diagnosis and hence to more adequate therapy. Originally he was concerned with the problem of whether or not a hearing loss was present; thus, his early tests were those which measured acuity, or the degree to which a given patient's responses to sound varied from a normal standard. The threshold of hearing was later measured as the amount by which sound had to exceed a normal threshold intensity for a given listener to hear. The otologist was next concerned, because of the widely differing medical and surgical rehabilitation needs, with the type of hearing loss present. This led to the differential diagnostic tests for conductive and nerve type impairments. Tests designed to satisfy the above mentioned needs constitute the bulk of audiometric examinations in clinical otological practice today. Methods and equipment for the measurement of discrimination ability for speech have been readily available to the clinician for several years; however, it is apparent that he has been slow to grasp the basic clinical importance of these discrimination tests. Such discrimination tests and test equipment in the form of turntable and records, which can be used satisfactorily in any office, have been waiting for general clinical acceptance.

It is not the intent of this paper to dwell on either the need

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for, or the technique of, various hearing tests which have both been abundantly covered in the literature. Rather, in reviewing the progress of hearing testing, emphasis will be placed on the development of those audiological tests which we believe to have *basic clinical application.*

Adam Politzer,[1] before the turn of this Century, recognized that non-calibrated tests such as the coin click, watch tick, Galton's whistle, whisper and spoken voice, and tuning fork tests "were insufficient as a means for ascertaining the exact condition of the function of hearing in diseases of the ear." It was recognized that these tests produced much variation in results as administered by different clinicians, with different standards. It was this variability which the inventors of the modern audiometer sought to eliminate.

TESTS OF ACUITY.

a. Tuning Fork Tests.

Tuning forks, as they were first used for measuring air conduction acuity, gave some idea of the sensitivity of the ear by measuring how long the patient continued to hear the tone. The use of forks representing each octave in the frequencies important for speech hearing, made possible for the first time an approximate plotting of the hearing threshold over a wide frequency spectrum. The serious limitation in the use of tuning forks as tests of acuity was the uncertainty as to the intensity of the sound produced.

b. Pure Tone Audiometric Tests.

Pure tone audiometric tests, by air conduction, represented a real advance in the precision of the measurement of hearing loss and the reliability of the results. The threshold of hearing was measured as the amount by which sound had to exceed a normal threshold intensity in order for a given patient to hear. While this test still serves us as a measurement of acuity for pure tones, or the *degree* of the hearing loss, it is necessary that its limitations be realized. Much confusion has resulted until recent years, due to the tendency to interpret the patient's *practical communication handicap* in the light of the air conduction pattern (see Fig. 1). The in-

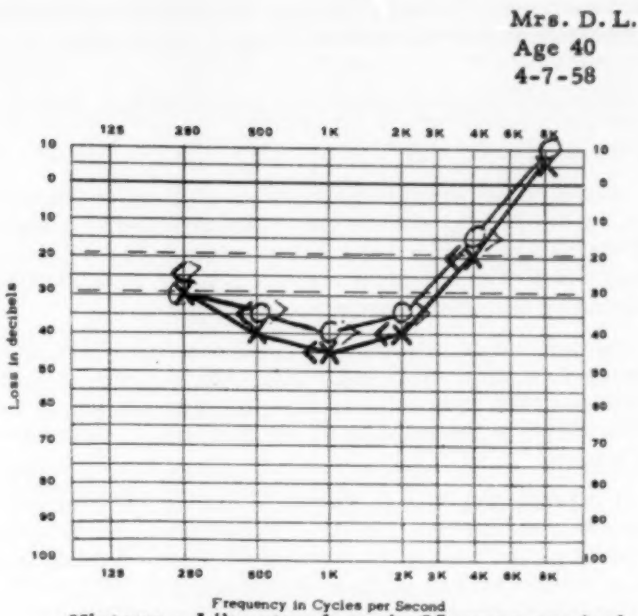


Fig. 1.

adequacy of the procedure alone became more apparent with the introduction of bone conduction testing and the development of speech perception tests.

c. Speech Reception Threshold.

In the course of investigation of the problem of communication in aircraft during World War II, Hudgins,[2] et al., developed the speech reception threshold tests as a measurement of acuity for speech. It was later established that the speech reception threshold usually correlates very closely with the pure tone average of 500, 1000, and 2000 c.p.s., representing the speech range, and thus it serves as a check on the accuracy

of the pure tone findings. The speech reception test has been adequately described numerous times in the literature.

DIFFERENTIATION OF CONDUCTIVE AND NERVE IMPAIRMENT.

a. Tuning Fork Tests.

Weber, Rinné and Schwabach[3] were pioneers in the use of the tuning fork as an instrument for differential diagnosis of the type of hearing impairment present. The well-known tests which they developed made it possible to differentiate conductive impairments from perceptive losses.

Politzer[1] wisely predicted the continued importance of the tuning fork as a differential diagnostic instrument in his textbook: "We most emphatically state that the tuning fork tests will be of lasting value in the diagnosis of disturbances of hearing."

It is interesting to note that all of our clinically applicable pure tone audiometric tests have as their basis the principles established in these early tuning fork tests. Tuning forks were further refined in their application and interpretation for making more specific diagnoses of types of hearing impairments or degrees of nerve involvement. This proved to be important in the selection of candidates for surgical rehabilitation.

I will clarify this particular subject with an illustration from our "Otology Study" sheet (see Fig. 2). This affords a graphic method of recording the results of tuning fork tests. From the shape and the position of the resultant curves, one can make both a rapid assessment of the patient's hearing status and, equally important, check on the accuracy of the bone conduction audiometric findings.

b. Bone Conduction Audiometry.

Bone conduction audiometry developed as a means of applying the methods of tuning fork testing in a more accurate and controlled fashion. With the introduction of the bone conduction oscillator, the intensity of the test tone could be calibrated, regulated and maintained at a constant level. In

spite of the improvement in the precision in generating the tone, it was soon found, as in the use of the tuning fork, that experience was essential in the application of the tests before any degree of confidence could be placed in the test results. It was all too often assumed that bone conduction testing was both easy and routine; however, problems such as placement

OTOLOGY STUDY SHEET

LEFT EAR

WEBER
250
500 →
1000 →

RINNE
- 250 -
▪ 500 -
⊣ 1000 -

RIGHT EAR

Mixed Loss

Pure conductive loss

250	500	1000	2000	SCHWABACH	250	500	1000	2000
X				Prolonged	X	X	X	
				Slightly prolonged				
				Normal				X
	X			Slightly shortened				
		X		Moderately shortened				
			X	Much shortened				
				Scarcely heard				
				Not heard				

	32	64	LOW FREQUENCY FORKS	32	64
			Normal		
			Slightly shortened		
	X		Much shortened		
			Scarcely heard		
		X	Not heard	X	X

Fig. 2.

of the oscillator, confused localization of the test tone, cross-over, and masking, made it a highly individualized and often challenging testing problem. It has proven to be the most important *pure tone* audiometric test, now clinically available, for the differential diagnosis of hearing impairment.

c. Above Threshold Pure Tone Tests.

The recruitment phenomenon was first described in 1924 by Pohlman and Kranz[4] and shortly after by Fowler,[5] in

1928. It was used to differentiate conductive and nerve type impairments; however, so many other tests more clearly demonstrated this diagnosis that they were given slight clinical acceptance.

It was only after Dix, Hallpike and Hood[6] demonstrated, in 1948, the usefulness of recruitment testing for the differential diagnosis of end-organ and VIIIth nerve impairments, that recruitment began to receive more general clinical attention.

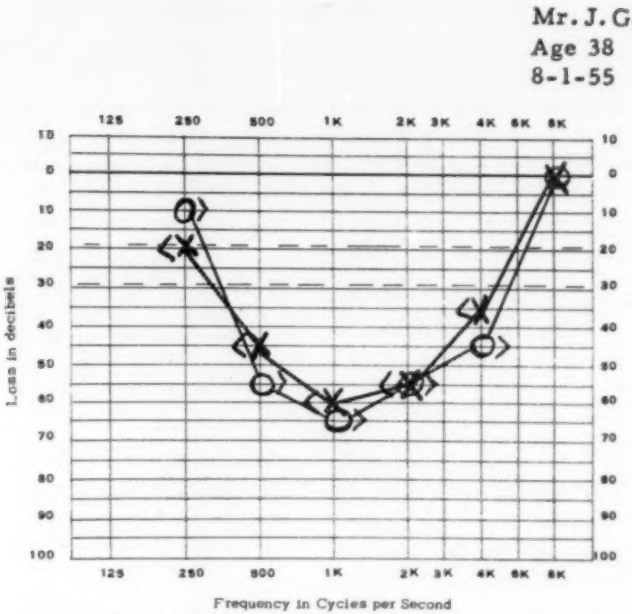
d. Speech Discrimination Tests.

The need for tests other than pure tone for the evaluation of a patient's hearing handicap has long been recognized. Oscar Wolfe,[7] in the latter part of the 19th Century said, that in order to make a differential diagnosis, it is absolutely necessary to use the *qualitative* test for speech hearing in addition to the *quantitative test* for hearing. MacFarland[8] described a number of early speech hearing tests dating back to 1929.

Egan,[9] in 1948, reported the development of various lists of phonetically balanced words to be used in the measurement of speech discrimination or intelligibility of speech. This led to the first practical clinical test for assessing the patient's *communication handicap* as distinguished from the more limited evaluation of the quantitative degree of his hearing loss (see Fig. 3).

MALINGERING TESTS.

Simulated deafness was widely recognized in the latter half of the 19th Century. A number of ingenious tests, employing tuning forks and stethoscopes along with careful observation of behavior, were used to diagnose non-organic hearing losses. During, and following World War II, the needs of the Armed Services prompted the widespread use and refinement of several audiometric tests for malingering, such as the Stenger, Lombard and Doerfler-Stewart. With the current active interest in industrial noise problems, including damage claims, it has become increasingly necessary for the otologist to distinguish the presence and extent of true organic hearing impairments from the non-organic or exaggerated overlay.



Limitations of air curve audiometry. Pure tone curves same, but wide difference in speech discrimination. Aid must be fitted to left ear.

Fig. 3.

TESTING THE VERY YOUNG CHILD.

A problem which has demanded increasingly more of the otologist's attention has been the peculiar testing needs associated with the assessment of hearing function in the very young child. This has necessitated the development of specialized motivational techniques and equipment for audiometry such as the Dix-Hallpike peep show[10] and the Pediacoumeter[11] (see Fig. 4); however, Mykelbust[12] and others[13] have pointed out that observation of behavior is often as important as hearing test results. This led to the use of behavioral inventories as an aid in the differential diagnosis of the delayed speech case with poor response to sound.

DIAGNOSIS OF END-ORGAN IMPAIRMENTS.

Another problem in otology has been to differentiate the site of neural involvement of hearing impairment, either in the receptor end-organ or in the transmission pathways, in order to provide a more complete diagnosis than was otherwise available. For this differential diagnosis, the presence of recruitment has been one of the most valuable indices.

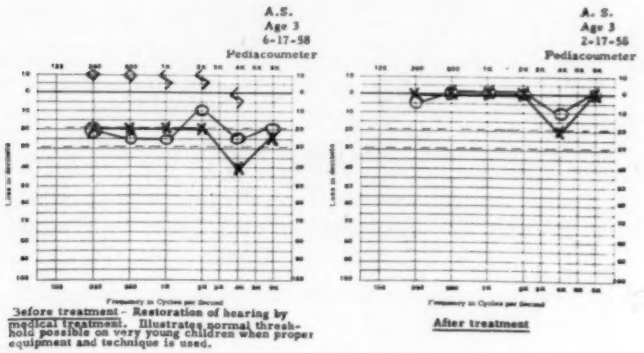


Fig. 4.

EVALUATION FOR AMPLIFICATION.

The otologist is now becoming increasingly aware of his responsibility to the patient who is not a candidate for surgical or medical rehabilitation. This has accentuated the need for taking various steps to evaluate the patient's requirements and suitability for amplification.[14,15] For this evaluation the most useful information audiologically has been that which comes from adequate air and bone audiometry, speech discrimination scores and recruitment testing (see Fig. 5).

The two most useful clinical tests for demonstrating recruitment have been the Fowler[5] binaural balance method and the monaural equal loudness method described by Watson.[16]

The binaural balance method has been limited in its application in that one non-recruiting ear must be present as a

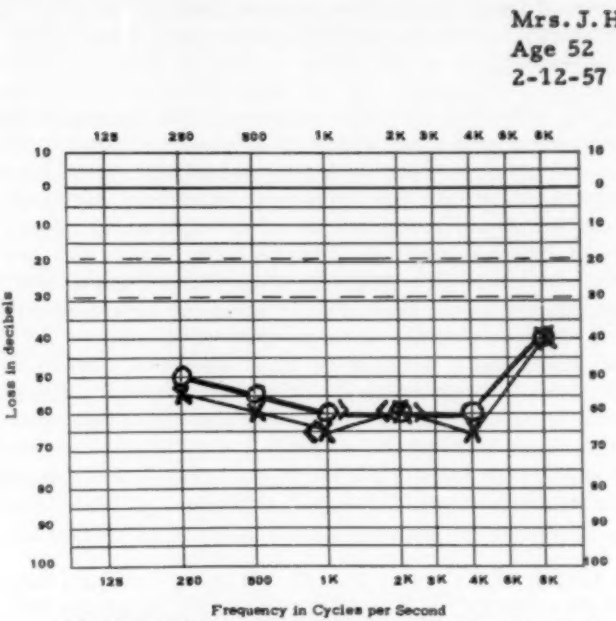


Fig. 5.

reference. The limitation in the monaural equal loudness test has been that a sloping threshold curve is necessary in order to demonstrate clearly convergence with the most comfortable equal loudness curve.

DISCUSSION.

In looking back on this review of clinical progress, it becomes necessary to ask ourselves—Where are we presently in the application of appropriate clinical hearing tests for adequate diagnosis? Do we have a practical group of clinical tests which may be economically applied for the accurate diagnosis of hearing problems? In the formulation of such

a group of tests, it should be stressed that not all of the old tests should be rejected and not all the new tests should as yet be accepted.

Of the tests used clinically, we believe that the tuning fork, air and bone conduction audiometry, speech perception tests, monaural equal loudness and binaural balance tests for recruitment, constitute the minimum battery of audiological tests which should be applied, as indicated, in a routine evaluation of hearing handicap. As we have pointed out previously,[17] facilities affording less than this complete hearing evaluation would not serve the situation for providing an accurate diagnosis and a rational plan of therapy. On the other hand, more exhaustive, time consuming evaluations with clinically unproven tests cannot be at present justified from a practical point of view. The basic audiological tests indicated can be adequately performed by trained personnel with moderately priced, commercially available clinical audiometers having a speech circuit or turntable. If the turntable with earphones is used, only a single quiet room is necessary.

A number of the newer laboratory tests of hearing function were not included in this discussion, since they have not as yet been established as practical clinical tools. We recognize, however, that the development and standardization of these tests in the laboratory have been of vital significance to the clinician.

Let us constantly and critically scrutinize the work of the laboratory in the development of new tests of hearing, in order to be prepared to supplant an outmoded test when a more reliable, more practical one appears; however, let us always keep in mind that the purpose of these tests is to provide an accurate diagnosis. If that is being adequately done with one or more current clinical tests, why should we feel that additional and often more complex ones are necessary, which at best would only provide confirmatory evidence? We have often allowed research bulletins and special electronic equipment representatives to lead us away from our basic clinical approach to the problem. The efforts of some of us to keep up with facilities for performing the very latest re-

ported tests has led not only to confusion in our specialty, but has also undermined the confidence of the majority of clinicians in their ability to arrive at an accurate clinical diagnosis. This has resulted in the widespread practice of either relegating this important clinical function to independent non-medical agencies, or to the rejection of all but the very simplest of air conduction audiometric or tuning fork screening tests, in an effort to evaluate the patient's communication handicap.

We firmly believe that the practical value of the routine application of the basic audiological tests mentioned has been adequately demonstrated, but that it needs to be more generally recognized clinically. Once the basic group of audiological tests is made thoroughly familiar to the practicing clinician and the resident in otolaryngology, as a minimum requirement for the evaluation of hearing function, real progress will be achieved, and we will enter a new era in the assessment of the patient's handicap in communication.

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Colby College, Waterville, Maine, presents the Sixth Annual Institute on Industrial Deafness, August 11-20 inclusive. Please note that the course has been extended for three days in order to include engineering for noise control. Its objective will be to train physicians interested in the problem; nurses, plant engineers and others in initiating and conducting hearing conservation programs in noisy industries. The course includes basic otology, audiometry, noise measurement, ear protection and methods of noise control. Class is limited to approximately twenty participants.

The fee for the course is $250 and includes tuition, room and board. The first week will constitute a complete course for doctors and nurses, and the reduced fee for this will be $200. Application should be made to Mr. William A. Macomber, Colby College, Waterville, Maine.

ADDENDA.

BASIC TECHNIQUES IN MYRINGOPLASTY AND TYMPANOPLASTY.

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Houston, Tex.

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Vice-President: Dr. Maurice Snitman, 408 So. 5th Ave., Maywood, Ill.
Secretary-Treasurer: Dr. Fletcher Austin, 700 No. Michigan Ave., Chicago 11, Ill.
Meeting: First Monday of each Month, October through May.

CHILEAN SOCIETY OF OTOLARYNGOLOGY.

President: Dr. Enrique Grünwald S.
Vice-President: Dr. Agustin Estartus.
Secretary: Dr. Marcos Chaimovich S.
Treasurer: Dr. Benjamin Kapkan K.
Director: Dr. Alberto Basterrica A.

DALLAS ACADEMY OF OPHTHALMOLOGY AND OTOLARYNGOLOGY

President: Dr. Ludwig A. Michael, 3707 Gaston Ave., Dallas, Tex.
Vice-President: Dr. Hal W. Maxwell.
Secretary-Treasurer: Dr. Edward A. Newell, 1511 No. Beckley, Dallas 8, Tex.

FEDERACION ARGENTINA, DE SOCIEDADES DE OTORRINOLARINGOLOGIA.

Secretary of the Interior: Prof. Dr. Atilio Viale del Carril.
Secretary of Exterior: Dr. Aldo G. Remorino.
Secretary Treasury. Prof. Dr. Antonio Carrascosa.
Pro-Secretary of the Interior: Prof. Dr. Carlos P. Mercandino.
Pro-Secretary of the Exterior: Prof. Dr. Jaime A. del Sel.
Pro-Secretary of the Treasury: Dr. Jorge Zubizarreta.

FIRST CENTRAL AMERICAN CONGRESS OF OTORHINOLARYNGOLOGY.

President: Dr. Victor M. Noubleau, San Salavador.
Secretary-Treasurer: Dr. Hector R. Silva, Calle Arce No. 84, San Salvador, El Salvador, Central America.

FLORIDA SOCIETY OF OPHTHALMOLOGY AND OTOLARYNGOLOGY.

President: Dr. Chas. C. Grace, 145 King St., St. Augustine, Fla.
President-Elect: Dr. Jos. W. Taylor, 706 Franklin St., Tampa, Fla.
Secretary-Treasurer: Dr. Carl S. McLemore, 1217 Kuhl Ave., Orlando, Fla.

FOURTH LATIN-AMERICAN CONGRESS OF OTORINOLARINGOLOGIA.

President: Dr. Dario.
Secretary:
Meeting:

GREATER MIAMI EYE, EAR, NOSE AND THROAT SOCIETY.

President: Dr. William B. Steinman.
President-Elect: Dr. James H. Mendel, Jr.
Secretary-Treasurer: Dr. H. Carlton Howard.
Meeting quarterly (March, May, October and December), on the second Thursday of the month, 6:30 P.M. at Urmey Hotel, Miami.

INTERNATIONAL BRONCHOESOPHAGOLOGICAL SOCIETY.

President: Dr. Jo Ono, Tokyo, Japan.
Secretary: Dr. Chevalier L. Jackson, 3401 N. Broad St., Philadephia 40, Pa., U. S. A.
Meeting: Seventh International Congress of Bronchoesophagology, Kyoto, Japan, September, 1958.

KANSAS CITY SOCIETY OF OTOLARYNGOLOGY AND OPHTHALMOLOGY.

President: Dr. Clarence H. Steele.
President-Elect: Dr. Dick H. Underwood.
Secretary: Dr. James T. Robison, 4620 J. C. Nichols Parkway, Kansas City, Mo.
Meeting: Third Thursday of November, January, February and April.

LOS ANGELES SOCIETY OF OPHTHALMOLOGY AND OTOLARYNGOLOGY.

President: Dr. Max E. Pohlman.
Secretary-Treasurer: Dr. Wendell C. Irvine.
Chairman of Ophthalmology Section: Dr. Carroll A. McCoy.
Secretary of Ophthalmology Section: Dr. Philip D. Shanedling.
Chairman of Otolaryngology Section: Dr. Robert W. Godwin.
Secretary of Otolaryngology Section: Dr. Francis O'N. Morris.
Place: Los Angeles County Medical Association Bldg., 1925 Wilshire Blvd., Los Angeles, Calif.
Time: 6:30 P.M. last Monday of each month from September to June, inclusive—Otolaryngology Section. 6:30, first Thursday of each month from September to June, inclusive—Ophthalmology Section.

LOUISIANA-MISSISSIPPI OPHTHALMOLOGICAL AND OTOLARYNGOLOGICAL SOCIETY.

President: Dr. Fred D. Hollowell, Lamar Life Bldg., Jackson, Miss.
Secretary: Dr. Edley H. Jones, 1301 Washington St., Vicksburg, Miss.
Meeting: Edgewater Gulf Hotel, Edgewater Park, Miss., May 15-16, 1959.

MEMPHIS SOCIETY OF OPHTHALMOLOGY AND OTOLARYNGOLOGY.

Chairman: Members serve as chairmen in alphabetical order monthly.
Secretary-Treasurer: Dr. Roland H. Myers, 1720 Exchange Bldg., Memphis, Tenn.
Assistant Secretary-Treasurer: Dr. William F. Murrah, Jr., Exchange Bldg., Memphis, Tenn.
Meeting: Second Tuesday in each month at 8:00 p.m. at Memphis Eye, Nose and Throat Hospital.

MEXICAN ASSOCIATION OF PLASTIC SURGEONS.

President: Dr. Cesar LaBoide, Mexico, D. F.
Vice-President: Dr. M. Gonzales Ulloa, Mexico, D. F.
Secretary: Dr. Juan De Dios Peza, Mexico, D. F.

MISSISSIPPI VALLEY MEDICAL SOCIETY.

President: Dr. Arthur S. Bristow, Princeton, Mo.
Secretary-Treasurer: Dr. Harold Swanberg, Quincy, Ill.
Assistant Secretary-Treasurer: Dr. Jacob E. Reisch, Springfield, Ill.

NETHERLANDS SOCIETY OF OTO-RHINO-LARYNGOLOGY.
(Nederlandsche Keel-Neus-Oorheelkundige Vereeniging.)

President: Dr. H. Navis, Sonsbeekweg 6, Arnhem.
Secretary: Dr. W. H. Struben, J. J. Viottastraat 1, Amsterdam.
Treasurer: Mrs. F. Velleman-Pinto, Jac. Obrechtstr. 66, Amsterdam.

NORTH CAROLINA EYE, EAR, NOSE AND THROAT SOCIETY.

President: Dr. J. C. Peele, Kinston Clinic, Kinston, N. C.
Vice-President: Dr. George E. Bradord, Winston-Salem, N. C.
Secretary-Treasurer: Dr. J. D. Stratton, 1012 Kings Drive, Charlotte 7, N. C.
Meeting:

NORTH OF ENGLAND OTOLARYNGOLOGICAL SOCIETY.

President: Mr. G. L. Thompson, 16 Ramshill Road, Scarborough, Yorkshire.
Vice-President: Mr. J. H. Otty, Frizley Old Hall, Frizinghall Road, Bradford, Yorkshire.
Secretary and Treasurer: Mr. R. Thomas, 27 High Petergate, York, Yorkshire.

OTOSCLEROSIS STUDY GROUP.

President: Dr. L. R. Boies, University Hospital, Minneapolis, Minn.
Secretary-Treasurer: Dr. Arthur L. Juers, 611 Brown Bldg., Louisville, Ky.
Meeting: Palmer House, Chicago, Ill.

PACIFIC COAST OTO-OPHTHALMOLOGICAL SOCIETY.

President: H. Leroy Goss, M.D., 620 Cobb Bldg., Seattle 1, Washington.
Secretary-Treasurer: Homer E. Smith, M.D., 508 East South Temple, Salt Lake City, Utah.
Meeting:

PAN AMERICAN ASSOCIATION OF OTO-RHINO-LARYNGOLOGY AND BRONCHO-ESOPHAGOLOGY.

President: Dr. Jose Gros, Havana, Cuba.
Executive Secretary: Dr. Chevalier L. Jackson, 3401 N. Broad St., Philadelphia 40, Pa., U. S. A.
Meeting: Sixth Pan American Congress of Oto-Rhino-Laryngology and Broncho-Esophagology.
Time and Place: Brazil, 1953.

PHILADELPHIA LARYNGOLOGICAL SOCIETY.

President: Dr. Chevalier L. Jackson.
Vice-President: Dr. John J. O'Keefe.
Treasurer: Dr. Joseph P. Atkins.
Secretary: Dr. Louis E. Silcox.
Historian: Dr. Herman B. Cohen.
Executive Committee: Dr. Harry P. Schenck, Dr.; Benjamin H. Shuster, Dr. William A. Lell, Dr.; William J. Hitschler.

PITTSBURGH OTOLOGICAL SOCIETY.

President: Dr. Bernard L. Silverblatt, 3500 Fifth Avenue, Pittsburgh, Pa.
Vice-President: Dr. Emory A. Rittenhouse, 203 Masonic Bldg., McKeesport, Pa.
Secretary-Treasurer: Dr. John T. Dickinson, Mercy Hospital, Pittsburgh 19, Pa.

PORTUGUESE OTORHINOLARYNGOLICAL SOCIETY.

President: Dr. Albert Luis de Mendonca.
Secretary: Dr. Antonio da Costa Quinta, Avenida, de Liberdale 65, 1° Lisbon.

PUGET SOUND ACADEMY OF OPHTHALMOLOGY AND OTOLARYNGOLOGY

President: Dr. Clifton E. Benson, Bremerton, Wash.
President-Elect: Dr. Carl D. F. Jensen, Seattle, Wash.
Secretary: Dr. Willard F. Goff, 1215 Fourth Ave., Seattle, Wash.

RESEARCH STUDY CLUB OF LOS ANGELES, INC.

Chairman: Dr. Orrie E. Ghrist, 210 N. Central Ave., Glendale, Calif.
Treasurer: Dr. Norman Jesberg, 500 So. Lucas Ave., Los Angeles 17, Calif.
Otolaryngology: Dr. Russell M. Decker, 65 N. Madison Ave., Pasadena 1, Calif.
Ophthalmology: Dr. Warren A. Wilson, 1930 Wilshire Blvd., Los Angeles 57, Calif.
Mid-Winter Clinical Convention annually, the last two weeks in January at Los Angeles, Calif.

SECTION OF OTOLARYNGOLOGY OF THE MEDICAL SOCIETY OF THE DISTRICT OF COLUMBIA.

Chairman: Dr. J. L. Levine.
Vice-Chairman: Dr. Russell Page.
Secretary: Dr. James J. McFarland.
Treasurer: Dr. Edward M. O'Brien.
Meetings are held the second Tuesday of September, November, January, March and May, at 6:30 P.M.
Place: Army and Navy Club, Washington, D. C.

SCOTTISH OTOLARYNGOLOGICAL SOCIETY.

President: E. A. M. Connal, 1 Royal Crescent, Glasgow C. 3, Scotland.
Secretary-Treasurer: Dr. J. F. Birrell, 14 Moray Place, Edinburgh.
Assistant Secretary: Dr. H. D. Brown Kelly, 11 Sandyford Place, Glasgow.

SOCIEDAD COLUMBIANA DE OFTALMOLOGIA Y OTORRINOLARINGOLOGIA (BOGOTA, COLUMBIA).

Presidente: Dr. Alfonso Tribin P.
Secretario: Dr. Felix E. Lozano.
Tesorero: Dr. Mario Arenas A.

SOCIEDAD CUBANA DE OTO-LARINGOLOGIA.

President: Dr. Reinaldo de Villiers.
Vice-President: Dr. Jorge de Cárdenas.
Secretary: Dr. Pablo Hernandez.

SOCIEDAD DE ESTUDIOS CLINICOS DE LA HABANA.

Presidente: Dr. Frank Canosa Lorenzo.
Vice-Presidente: Dr. Julio Sanguily.
Secretario: Dr. Juan Portuondo de Castro.
Tesorero: Dr. Luis Ortega Verdes.

SOCIEDAD DE OTORRINOLARINGOLOGIA Y BRONCOESOFAGOSCOPIA DE CORDOBA.

Presidente: Dr. Aldo Remorino.
Vice-Presidente: Dr. Luis E. Olsen.
Secretario: Dr. Eugenio Romero Diaz.
Tesorero: Dr. Juan Manuel Pradales.
Vocales: Dr. Osvaldo Suárez, Dr. Nondier Asis R., Dr. Jorge Bergallo Yofre

SOCIEDAD DE OTO-RINO-LARINGOLOGIA, COLEGIO MEDIO DE EL SALVADOR, SAN SALVADOR, C. A.

President: Dr. Salvador Mixco Pinto.
Secretary: Dr. Daniel Alfredo Alfaro.
Treasurer: Dr. Antonio Pineda M.

SOCIEDAD ESPANOLA DE OTORRINOLARINGOLOGIA.

Presidente: Dr. D. Adolfo Hinojar Pons.
Vice-Presidente: Dr. D. Jose Perez Mateos.
Secretario General: Dr. D. Francisco Marañés.
Tesorero: Dr. D. Ernesto Alonso Ferrer.

SOCIEDAD MEXICANA DE OTORRINOLARINGOLOGIA

Havre 7—Desp. 62
Mexico 6, D. F.

Honorary President: Dr. Ricardo Tapia y Fernández.
President: Dr. Máximo García Castañeda.
Secretary: Dr. Eduardo de la Parra.
Treasurer: Dr. Guillermo Pérez Villasante.
Vocal: Dr. Rafael Pacchiano.

SOCIEDAD NACIONAL DE CIRUGIA OF CUBA.

Presidente: Dr. Reinaldo de Villers.
Vice-Presidente: Dr. César Cabrera Calderin.
Secretario: Dr. José Xirau.
Tesorero: Dr. Alfredo M. Petit.
Vocal: Dr. José Gross.
Vocal: Dr. Pedro Hernández Gonzalo.

SOCIEDAD OTO-RINO-LARINGOLOGIA DE LOS HOSPITALES DE MADRID.

Presidente: Dr. Don Fernando Beltrán Castillo.
Secretario General: Dr. Don Alfonso Vassallo de Mumbert.
Tesorero: Dr. Don Rafael García Tapia.

SOCIEDAD VENEZOLANA DE OTORRINOLARINGOLOGIA.

Presidente: Dr. Alfredo Celis Pérez.
Vice-Presidente: Dr. Bustamante Miranda.
Secretario General: Dr. Jesús Miralles.
Tesorero: Dr. M. Matheus.
Vocales: Dr. Perez Velasquez and Dr. Wilmer Palacios.

SOCIEDADE DE OFTALMOLOGIA E OTORRINOLARINGOLOGIA DO RIO GRANDE DO SUL.

President: Dr. Paulo Fernando Esteves.
Vice-President: Dr. Jayme Schilling.
First Secretary: Dr. Carlos Buede.
Second Secretary: Dr. Moizés Sabani.
First Treasurer: Dr. Israel Scherman.
Second Treasurer: Dr. Rivadávia C. Meyer.
Librarian: Dr. Carlos M. Carrion.

SOCIEDAD PANAMENA DE OTORRINOLARINGOLOGIA

Presidente: Dr. Manuel Preciado.
First Vice-Presidente: Dr. Alonso Roy.
Second Vice-Presidente: Dr. Carlos Arango Carbone.
Secretario: Dr. María Esther Villalaz.
Tesorero: Dr. Ramòn Crespo.

SOCIEDADE PORTUGUESA DE OTORRINOLARINGOLOGIA E DE BRONCO-ESOFAGOLOGIA.

Presidente: Dr. Alberto Luis De Mendonca.
Vice-Presidente: Dr. Jaime de Magalhaes.
1.° Secretario: Dr. Antonio da Costa Quinta.
2.° Secretario: Dr. Albano Coelho.
Tesoureiro: Dr. Jose Antonio de Campos Henriques.
Vogais: Dr. Teofilo Esquivel.
Dr. Antonio Cancela de Amorim.
Sede: Avenida da Liberdade, 65, 1°, Lisboa.

SOCIETY OF MILITARY OTOLARYNGOLOGISTS.

President: Capt. William C. Livingood, U.S.N. (M.C.)
Secretary-Treasurer: Lt. Col. Sanley H. Bear, M.C., 3310th USAF Hospital, Maxwell AFB, Alabama.
Meeting: Palmer House, Chicago, Ill.

SOUTH CAROLINA SOCIETY OF OPHTHALMOLOGY AND OTOLARYNGOLOGY.

President: Dr. James H. Gressette, Orangeburg, S. C.
Vice-President: Dr. Robert P. Jeanes, Easley, S. C.
Secretary-Treasurer: Dr. Roderick Macdonald, 333 East Main St., Rock Hill, S. Car.
Meeting:

SOUTHERN MEDICAL ASSOCIATION, SECTION ON OPHTHALMOLOGY AND OTOLARYNGOLOGY.

Chairman: Dr. V. Eugene Holcombe, Charleston, W. Va.
Chairman-Elect: Dr. G. Slaughter Fitz-Hugh, Charlottesville, Va.
Vice-Chairman: Dr. George M. Haik, New Orleans, La.
Secretary: Dr. Mercer G. Lynch, New Orleans, La.

VIRGINIA SOCIETY OF OPHTHALMOLOGY AND OTOLARYNGOLOGY.

President: Dr. Benjamin Sheppard, 301 Medical Arts Building, Richmond, Virginia.
President-Elect: Dr. Emanuel U. Wallerstein, Professional Building, Richmond, Virginia.
Vice-President: Dr. Calvin T. Burton, Medical Arts Building, Roanoke, Virginia.
Secretary-Treasurer: Dr. Maynard P. Smith, 600 Professional Building, Richmond, Virginia.
Meeting:

WEST VIRGINIA ACADEMY OF OPHTHALMOLOGY AND OTOLARYNGOLOGY.

President: Dr. James K. Stewart, Wheeling, W. Va.
Secretary-Treasurer: Dr. Frederick C. Reel, Charleston, W. Va.
Annual Meeting: